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THE MECHANISM OF THE HEART BEAT.

THE MECHANISM OF THE HEART BEAT

WITH ESPECIAL REFERENCE TO ITS
CLINICAL PATHOLOGY

BY

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TO
JAMES MACKENZIE
AND
WILLEM EINTHOVEN

A TOKEN OF APPRECIATION
OF THE SERVICES WHICH THEY HAVE RENDERED
TO THE STUDY OF THE
CLINICAL PATHOLOGY OF HEART AFFECTIONS.

PREFACE.

OUR knowledge of the clinical pathology of the heart has advanced with such rapidity during the last decade, and the subject as a whole has become so technical, that at the present time it is difficult or well-nigh impossible for the general reader to keep pace with its progress. The writings are scattered; they call, in the reading, for considerable preliminary knowledge of the subject matter discussed.

In contemplating the work of the past few years, it seemed to me that a stage of the enquiry had been reached at which it was possible to give a review of the main results of the numerous researches, and to place before the student of general medicine the evidences upon which the chief conclusions of the present day rest.

A number of phenomena, observed in clinical and experimental studies, are described side by side in this book, and an attempt is made to show the manner in which abnormal actions of the heart, as they occur in patients, may be identified with similar disturbances artificially created in laboratory experiment.

It has been deemed advisable to restrict the chapters, so far as possible, to a discussion of those subjects alone, in which our knowledge of the facts and the conclusions derived therefrom appear to stand on a sure footing.

It is for this reason that the reader will find no reference in the following pages to the still controversial questions of the

“neurogenic” or “myogenic” origin and transmission of the heart beat. The walls of the heart are composed of a syncytium of muscle fibres, closely interwoven with nerve fibrils and ganglia; in speaking of the musculature of the heart, I have done so upon the distinct understanding that I speak of muscle in full functional connection with the nerve elements surrounding it. For, dealt with in this manner, it is immaterial to the subjects considered and to the conclusions arrived at, whether one or other view is held.

It may not be inappropriate to refer at this time to a general terminology employed by writers upon disorders of the cardiac mechanism and especially to certain terms which are used to denote certain physiological properties of cardiac muscle. The strict separation of five cardiac functions, rhythmicity, excitability, contractibility, conductivity, and tonicity, is one which, as I am well aware, is jealously guarded by many writers, more especially the disciples of Engelmann. There may or may not be justification for this doctrine. Be that as it may, the emphasis laid upon the isolation of separate functions is found by experience to form but an insecure foundation for the classification of cardiac irregularities. Therefore, in using the convenient terms *rhythmicity* and *conductivity*, or their derivatives, in the succeeding chapters, I employ them only in so far as they indicate the observed facts, the origin of heart beats in a limited area and in rhythmic fashion, the transmission of waves of contraction from one chamber to another, or from one portion of the musculature to another.

The forms of disordered mechanism to which the human heart is subject are numerous; but the majority of clinical examples are of comparatively simple form and fall into a few distinct and well defined categories. Instances of more complex disturbances remain,

which when encountered must be treated largely upon their individual merits. Such disturbances receive but little detailed attention in this book, for they concern and always must concern the special worker more particularly. Their consideration would not only have necessitated a considerable expansion of the work, but it would have endangered a burial of the broader principles in a mass of detail which many could not have failed to discover tedious if not irksome.

The nature of the subject necessitates abundant reference to graphic records, of which I have endeavoured to give examples in which the analysis will be unquestioned by those familiar with the methods. For the sake of simplicity, the text deals with electrocardiographic curves obtained by means of a single and customary lead.

Of the immediate value of graphic methods in the practice of medicine, it is my desire to speak but briefly. They have placed the entire question of irregular or disordered mechanism of the human heart upon a rational basis, so giving to the physician the confidence of real knowledge; they have profoundly influenced and have added exactitude to prognosis; they have potentially abolished the promiscuous exhibition of cardiac poisons, and have clearly shown the lines which their administration should follow. The new clinical observations have stimulated and directed a host of laboratory researches, anatomical, physiological, pathological, and pharmacological, of the most valuable nature.

The records in themselves constitute the most exact signs of cardiac affections which we possess. The little strips of paper, imprinted by the disease itself, form permanent and unquestionable testimony of events which have occurred, and may be placed in the balance, without disquietude, while experiences of a more subjective kind fill the opposing scale.

I have written in the hope of stimulating and aiding those who are commencing the study of the heart by modern means, and in the hope of supplying the general physician or practitioner with an outline of new physiological and pathological knowledge, which may be of service to him in his wards or in his practice.

It is my pleasant duty to express my appreciation of the unfailing kindness of my friend Dr. James Mackenzie, with whom it has been my privilege to have been closely associated during the past few years.

I have to thank a number of friends and the publishers of several periodicals for permission to publish or republish certain of the curves. The detailed acknowledgments are conveyed in the explanatory notes which accompany the figures in question. I have also to thank Henry Frowde, Esq., and Edward Arnold, Esq., publishers, for their courtesy in allowing the republication of paragraphs of the text of original papers.

For his careful revision and correction of the proofs I am under obligations to the kindness of my friend and colleague Dr. T. R. Elliott.

It is my final duty, to acknowledge indebtedness to the Beit Memorial Research Fund for the leisure in which to write, and to state that many of the hitherto unpublished observations were made under the tenure of a Fellowship provided by it.

Th. L.
58a, Wimpole Street,
December, 1910.

CONTENTS.

CHAPTER	PAGE
I.—RECENT ANATOMICAL DISCOVERIES	1
<i>The sino-auricular node</i>	2
<i>The auriculo-ventricular connection</i>	2
II.—INTRA-AURICULAR PRESSURE CURVES AND VENOUS RECORDS	9
<i>Intra-auricular pressure variations in experiment</i> ..	9
<i>The venous pulse in animals and man</i>	9
<i>The cardiac events upon which the waves of the auricular pressure and jugular pulse curves depend</i>	13
<i>The first positive or “a” wave</i>	13
<i>The second positive or “c” wave</i>	15
<i>The third positive or “v” wave</i>	15
<i>The first and second negative waves or “x” and “x’” depressions</i>	16
<i>The third negative wave or “y” depression</i> ..	16
III.—THE GALVANOMETRIC METHOD AND THE PHYSIO- LOGICAL ELECTROCARDIOGRAM	18
<i>The string galvanometer</i>	18
<i>The physiological electrocardiogram</i>	21
<i>The leads adopted</i>	21
<i>The physiological type of mammalian electro- cardiogram</i>	23
<i>The chief time relationships of the electrocardio- gram</i>	24
<i>The meaning of the electrocardiogram</i>	25
IV.—THE PACE-MAKER OF THE MAMMALIAN HEART	31
<i>The electric changes accompanying the auricular con- tractions, normal and excited</i>	32
<i>The point of primary negativity</i>	36
<i>Other observations</i>	40
<i>Observations upon the dying heart</i>	40
<i>Warming and cooling the sinus</i>	40
<i>Excision or section</i>	40
<i>The length of the pause succeeding artificially excited beats</i>	41

V.—THE ANALYSIS OF THE CARDIAC MECHANISM

ARTERIAL PULSE CURVES 43

<i>The presence or absence of a rhythm dominating the movements of the ventricle</i>	44
<i>The presence of regular sequences of beats</i>	46
<i>Phasic variation of the dominant rhythm</i>	46
<i>The rate of the dominant rhythm</i>	47
<i>The presence or absence of disturbance of the dominant rhythm at its source</i>	47
<i>The presence or absence of interference with the dominant impulses during their passage to the ventricle</i>	48
<i>The determination of the length of the pauses following single or successive premature beats</i>	49
<i>Complete irregularity of the pulse</i>	49

VI.—THE ANALYSIS OF THE CARDIAC MECHANISM

POLYGRAPHIC CURVES 51

<i>The fixation of "c" in the jugular curve</i>	51
<i>The fixation of "a" in the jugular curve</i>	53
<i>The "As-Vs" interval</i>	53
<i>The value of the fixation of the summit of "v"</i>	53
THE INTERPRETATION OF CURVES IN WHICH THERE ARE	
DISTURBANCES OF SEQUENCE	55
<i>Prominent peaks</i>	56
<i>Supernumerary "a" waves</i>	60
<i>Venous accompaniments of premature beats</i>	60
<i>Absence of "a" waves from the jugular pulse</i>	62

VII.—THE ANALYSIS OF THE CARDIAC MECHANISM

ELECTROCARDIOGRAPHIC CURVES .. 65

<i>The recognition of auricular and ventricular contractions of physiological type</i>	65
<i>Ventricular complexes which are of supraventricular origin</i>	65
<i>The identification of beats which arise in the vicinity of the pace-maker</i>	66
<i>The identification of beats which belong to the normal rhythm</i>	66
<i>The recognition of auricular and ventricular contractions of anomalous type</i>	67
<i>Variations which are encountered in the outline of the electrocardiogram when the heart is regular and the normal sequence is present</i>	70

VII.—THE ANALYSIS OF THE CARDIAC MECHANISM ELECTROCARDIOGRAPHIC CURVES

(continued)

<i>The auricular complex especially associated with hypertrophy of the auricle</i>	72
<i>Alterations in the magnitude and direction of the variation " T " of the ventricular complex</i> ..	72
<i>The algebraic summation of complexes</i>	74

VIII.—EXPERIMENTAL HEART - BLOCK IN THE MAMMAL 79

<i>The evidence that the auriculo-ventricular bundle transmits the impulses from auricle to ventricle in the mammalian heart</i>	79
<i>Heart-block as a result of other interferences</i>	84
<i>Records of experimental heart-block</i>	86

IX.—CLINICAL HEART-BLOCK 89

<i>The signs of heart-block in man</i>	89
<i>The nature of alternating 1 : 1, 2 : 1 and 2 : 1, 3 : 1 ratios</i>	93

X.—THE CAUSATION OF CLINICAL HEART-BLOCK .. 99

<i>The post-mortem findings</i>	99
<i>The relationship of heart-block to rheumatic infection of the heart and mitral stenosis</i>	107
<i>The action of drugs of the digitalis group upon heart-block</i>	108
<i>The vagus as a cause of clinical heart-block</i>	108

XI.—THE IDIO-VENTRICULAR RHYTHM ; HOMOGENETIC AND HETEROGENETIC HEART BEATS 113

<i>The idio-ventricular rhythm</i>	113
<i>Homogenetic heart contractions</i>	117
<i>Heterogenetic contractions and their contrast with homogenetic beats</i>	117

XII.—SINGLE HETEROGENETIC HEART CONTRACTIONS 122

<i>The premature ventricular contraction</i>	122
<i>The ways in which premature contractions are evoked in the ventricle</i>	126
<i>The recognition of premature ventricular contractions in clinical subjects</i>	126
<i>The premature auricular contraction</i>	128
<i>The recognition of premature auricular beats in man</i>	132
<i>Premature beats arising in the junctional tissues</i> ..	132
<i>The recognition of premature junctional beats in man</i>	136

XIII.—SINGLE HETEROGENETIC HEART CONTRACTIONS

(continued)	139
<i>The electric changes accompanying premature auricular contractions</i>	139
<i>The ventricular complex which arises as a result of a premature auricular contraction</i>	140
<i>The electric waves accompanying premature beats, in which auricle and ventricle contract together</i>	146
<i>The electric curves given by premature contractions arising in the ventricle</i>	146

XIV.—HETEROGENETIC RHYTHMS (SUCCESSIVE PATHOLOGICAL CONTRACTIONS AND TACHYCARDIAS OF VENTRICULAR ORIGIN)

<i>The experimental production of heterogenetic rhythms</i> ..	156
<i>The production of heterogenetic rhythms in the ventricle</i>	158
<i>Heterogenetic rhythms arising in the ventricles of patients</i>	163

XV.—HETEROGENETIC RHYTHMS (continued) (SUCCESSIVE PATHOLOGICAL CONTRACTIONS AND TACHYCARDIAS OF AURICULAR ORIGIN)

<i>Clinical tachycardias of auricular origin</i>	172
<i>Example of transitory tachycardia of auricular origin</i>	180
<i>Example of tachycardia arising in the vicinity of the auriculo-ventricular node; nodal rhythm</i>	181
<i>General observations</i>	184

XVI.—PAROXYSMAL TACHYCARDIA (AN OBSCURE FORM; CLASSIFICATION OF TACHYCARDIAS; RELATIONSHIP TO FIBRILLATION)

<i>An obscure form of tachycardia (paroxysms of regular tachycardia accompanied by the ventricular form of venous pulse)</i>	186
<i>The classification of paroxysmal tachycardia</i>	187
<i>Terminal events in paroxysmal tachycardia; the appearance presented by the auricle in a state of fibrillation</i>	188

XVII.—CLINICAL AURICULAR FIBRILLATION	194
HISTORICAL	194
THE CLINICAL CONDITION	196
<i>The radial pulse curves</i>	196
<i>The venous pulse curves</i>	198

XVII.—CLINICAL AURICULAR FIBRILLATION (*continued*)

<i>Certain waves which occur in diastole</i>	201
<i>Electrocardiographic curves obtained by leads from the right arm and left leg</i>	204
COMPLETE IRREGULARITY OF THE HEART IS THE RESULT OF AURICULAR FIBRILLATION	207
<i>The irregular oscillations seen upon galvanometric curves are due to an inco-ordinate contraction of some portion of the heart; they are not a direct result of structural change in the heart, and are independent of movements of the somatic musculature</i>	207
<i>The irregular oscillations arise in the vicinity of the auricle; the ventricular electric complex in complete irregularity is of the normal form</i>	212
<i>Conclusions from the clinical findings</i>	213

XVIII.—AURICULAR FIBRILLATION (*continued*) 216

<i>Experimental and clinical electric curves compared</i> ..	216
<i>The electrocardiographic curves compared in more detail</i>	218
<i>The arterial curves compared</i>	223
<i>The venous curves and their comparison</i>	224
<i>A summary of the comparison instituted between the records obtained in complete irregularity of the heart in man and in experimental auricular fibrillation</i> ..	226
<i>Auricular fibrillation as a clinical entity</i>	228
<i>Auricular fibrillation in the horse</i>	230
<i>The morbid anatomy in clinical auricular fibrillation</i> ..	232

XIX.—THE ASSOCIATION OF TACHYCARDIA OF AURICULAR ORIGIN AND AURICULAR FIBRILLATION WITH HEART-BLOCK. THE ACTION OF DIGITALIS 234

<i>Acceleration of the auricle and heart-block</i>	234
<i>Auricular fibrillation and heart-block</i>	235
<i>Complete dissociation and auricular fibrillation</i>	237
<i>Lesser grades of heart-block and fibrillation</i> ..	239
<i>The effect of vagal stimulation upon auricular fibrillation</i>	239
<i>The action of digitalis in auricular fibrillation</i>	242

XX.—AURICULAR FIBRILLATION AND HETEROGENETIC BEATS OF VENTRICULAR ORIGIN. SO-CALLED 'BIGEMINY' 247

<i>Auricular fibrillation and heterogenetic beats of ventricular origin. The coupled beats of digitalis</i>	247
<i>So-called 'bigeminy' of the heart</i>	249

CHAPTER	PAGE
XXI.—THE VAGUS	253
<i>Respiratory arrhythmias</i>	253
<i>Respiratory arrhythmia in experiment</i>	253
<i>Respiratory arrhythmia in the healthy human subject</i>	253
<i>Respiratory arrhythmia, associated with pathological processes</i>	254
<i>Irregularities produced by vagal stimulation in animals</i>	257
(i). <i>Slowing</i>	257
(ii). <i>Standstill of the heart</i>	258
(iii). <i>Hindrance to the passage of auricular impulses to the ventricle</i>	258
(iv). <i>Escape of the ventricle</i>	258
<i>Examples of vagal phenomena as they occur clinically</i>	258
<i>Phasic irregularity independent of respiration</i>	258
<i>Compression of the vagus in the human subject</i>	259
<i>Standstill of the heart as a clinical phenomena</i> ..	259
<i>Escape of the ventricle in man</i>	261
<i>Complex conditions</i>	261
<i>General remarks</i>	262
 XXII.—THE SO-CALLED “ADAMS-STOKES’ SYNDROME” ..	265
<i>The cause of the syncopal and epileptic seizures</i> ..	265
<i>The cause of the ventricular slowing</i>	266
<i>The fits of partial heart-block</i>	268
<i>The fits of complete heart-block</i>	268
 XXIII.—ALTERNATION	271
<i>Clinical alternation</i>	271
<i>Alternation in experiment</i>	273
<i>The nature of alternation ; the conditions giving rise to it and its significance</i>	276
 INDEX	281
 PLATES—I—V	296

Fig. 1. A figure published through the kindness of Dr. Ivy Mackenzie. A section (magnification 330 diam.) taken through the *sulcus terminalis* of a pig's auricle and stained with hæmatoxylin and Van Gieson's stain. The plane of section is in the line of the sulcus and at right angles to the wall of the auricle. The epicardium lies to the left, the endocardium to the right. The muscle bundles of the auricular musculature are shown to the right, cut in length and across. To the left and beneath the epicardium is the dense collection of peculiar tissue of the sino-auricular node. It is compact, the muscle fibres small, interlaced and presenting numerous nuclei. A large vessel is seen on the outskirts of the tissue and numerous nerve fibres and ganglion cells are embedded in the node itself.



Fig. 1.

CHAPTER I.

RECENT ANATOMICAL DISCOVERIES.

RECENT work, consisting of a close examination of certain regions of the cardiac musculature by means of serial histological sections, has revealed the presence of certain specialised structures, an acquaintanceship with which is of fundamental importance to a study of the normal and disordered heart beat. They are briefly described in the following paragraphs.

It has been recognised for many years, and chiefly as a result of the work of Stannius, Gaskell and Engelmann, that the beat of the amphibian and reptilian heart has its origin in the *sinus venosus*, that portion of the organ into which the systemic veins flow, and which constitutes a separate and well-defined chamber in the lower vertebrates.

In the mammalian heart sinus and auricle are fused into one cavity, the anatomical auricle, and morphologists have attempted to define the limits of the original sinus tissue by studying the relationship of the entering veins in the several types of vertebrate heart. The older conception that the *sulcus terminalis*, a groove lying upon the superficial surface of the right auricle and extending from superior to inferior vena cava, is a representation of the sino-auricular junction can be maintained no longer; neither can it be held that a strict anatomical separation is still extant in the mammalian heart.

The view adopted by Keith⁷ is that the greater part of the tissue of the auricles and ventricles is an outgrowth from the primitive cardiac tube. In the auricle the new tissue has spread over the primitive remains, covering or displacing them. Tissue, which may be held to represent portions of the primitive canal in the auricle itself, is to be found in isolated masses at the superior cavo-auricular junction (a mass which has been termed the sino-auricular node) and in the vicinity of the coronary sinus; remnants of it appear to exist also in the auricular septum, in the valve of Eustachius and at the mouths of the pulmonary veins. The tissues uniting auricle and ventricle are regarded as belonging to the same system.

The importance of the conception that the sino-auricular node and the tissues joining auricle and ventricle are representatives of similar morphological structures will be more obvious at a later stage. It is based upon the fusiform character of the muscular fibres, of which they are largely composed, and their general structural similarity;* upon the positions

* Recent histological observation^{12 & 14} has added a further comparison between the two main collections, the sino-auricular node and the auriculo-ventricular bundle and its connections, namely that they both present a remarkable glycogen content.

which they occupy* and their morphological relationships; upon the probability that they constitute those portions of the muscle in which rhythms are most readily developed; and upon the intimate connection with nerve ganglia and networks.

The sino-auricular node.

Keith and Flack,⁷ while examining the musculature of the auricles, have happened upon what they regard as a special remnant of the original sinus tissue. It lies at the junction of the superior vena cava and right auricle. (The approximate position and longitudinal extent of this node is shown by means of a dotted line in Fig. 20.) "In the human heart, as in most mammalian hearts," these authors write, "an artery or arterial circle lies in the junction; the artery is surrounded by fibrous tissue in which are numerous peculiar muscle fibres, some nerve cells and some nerve fibres. The nerve cells and fibres we find from dissection to connect with the vagal and sympathetic nerve trunks." "Our search for a well-differentiated system of fibres within the sinus, which might serve as a basis for the inception of the cardiac rhythm, has led us to attach importance to this peculiar musculature surrounding the artery at the sino-auricular junction." "In the human heart the fibres are striated, fusiform, with well-marked elongated nuclei, plexiform in arrangement, and embedded in densely packed connective tissue—in fact, of closely similar structure to the Knoten" (referring to the auriculo-ventricular node to be described presently). These findings have been confirmed and extended by Koch,^{10 & 11} Schönberg^{16 & 17} and others. The special muscle system lies at the junction of the free border of the appendix with the superior caval termination, and extends downwards along the sulcus terminalis for a distance of about 2 cm. in man. In thickness it is approximately 2 mm.. The muscular fibres are small, being but a half or third the breadth of those of auricular fibres proper. This structure is termed the *sino-auricular node*, (Fig. 1).

As will be seen, the heart beat may be shown to arise in this node, and the contraction travels from it over the walls of the auricle and reaches the structures described in the following paragraphs.

The auriculo-ventricular connection.

The discovery of the anatomical connection between auricle and ventricle followed upon Wooldridge²¹ and Tigerstedt's¹⁹ demonstration of functional continuity in the mammal, and the elaborate researches of Gaskell,⁴ by which the dependence of the ventricular rhythm upon impulses derived from the auricle was clearly established in the cold-blooded heart.

* As Ivy Mackenzie has pointed out, the sino-auricular node is related to the right and the auriculo-ventricular node to the left duct of Cuvier,

The first, an imperfect account of muscular connections between auricle and ventricle in the mammal, was given by Kent^{8 & 9} in 1892. His preliminary paper was succeeded in 1893 by the publication of the independent observations of His (Jr.),⁵ who described a special bundle of muscle fibres running from auricle to ventricle. In 1904 Retzer¹⁵ and Braeunig¹ substantiated the foregoing observations, but the main advance was made by Tawara,¹⁸ working under the direction of Asehoff. In his book, *Das Reizleitungssystem des Säugetierherzens*, a complete account of the junctional tissues was given, and the anatomy of the whole system and the connections with the network of Purkinje were described in great detail and in many species of animals. These observations upon the anatomy have received complete confirmation by the more recent writings.^{2, 3, 6 & 13} All observers are agreed that one and a single path of anatomical communication exists between the upper and lower chambers of the heart.

The account which follows is based in the main upon the descriptions of Tawara and applies to the human heart.

The fibres of the junctional tissues may be traced from auricle to ventricle without break (Fig. 2). The system commences in the auricle in the neighbourhood of the coronary sinus and at the base of the auricular septum, where a collection of the auricular fibres ultimately joining a specialised structure, the *auriculo-ventricular node* is to be found. The node lies at the very edge of the auricular tissue at the posterior and right border of the septum. The bundle proper commences at the node, running almost horizontally forwards and to the left, ensheathed and isolated in a fibrous canal, and pursuing a course directly to the right of the central fibrous body of the heart to the *pars membranacea septi* of the ventricle. At the anterior part of this membrane the bundle divides, and the point of division lies a little in front of the anterior end of the attachment of the median or septal segment of the tricuspid valve to the ring. The left division of the bundle perforates the membrane, still lying ensheathed upon the upper border of the muscular septum, and enters the subendocardial space of the left ventricle at a point immediately beneath the union of the anterior and right posterior cusps of the aortic valve. Its further course is downward, and it may be traced under the endocardium of the septum of the left side. The right branch takes a subendocardial position directly after the division and, coursing downwards, enters the moderator band, or its representative, and proceeds directly to the papillary muscles where it commences its arborisation. The arborisation of the left division (Fig. 3) starts upon the septum, and the main branches flow to the papillary muscles of the mitral valve. The arborisation on the right and left side is directly continuous with the extensive and complex subendocardial network of *Purkinje fibres*, which lines the greater part of the interior of both ventricles. From this network direct communication with the ventricular muscle fibres takes place. The smaller ramifications and strands of the network itself are frequently carried across the valleys



Fig. 2. A specimen in the Royal College of Surgeons Museum, photographed with the kind permission of Professor Keith. A human heart seen from the front and right. The anterior walls of the right ventricle and right auricle have been removed. The intra-auricular septum, the tricuspid valve, the papillary muscles (*G*), the moderator band (*F*) and interior of the infundibulum (*H*) are exposed. *A* lies in the right auricular appendix. *B* lies in the *fossa ovalis*. *E* is placed below the mouth of the coronary sinus; directly to the right of it in the figure an area of endocardium has been removed and the upper connection of the auriculo-ventricular node with the musculature of the septum has been exposed. It consists of a fan-shaped piece of muscle which lies directly beneath *D*. A bristle has been placed beneath the fan. From this point the auriculo-ventricular bundle and its right branch are readily traced as they lie on a series of five bristles between *D* and *F*. The structure proceeds in a curved fashion to the membranous septum, which lies directly below *C*, and at this point the left branch passes through the septum. The right branch is continued upon the intraventricular septum and enters and follows the moderator band (*F*) until it reaches the base of the large group of papillary muscles (*G*). The arborisation which commences at this point is not clear in the photograph.



Fig. 3. A specimen in the Royal College of Surgeons Museum, photographed with the kind permission of Professor Keith. The heart of a walrus dissected from the left side. The greater portions of wall of the left ventricle and left auricle (A) have been removed and the aorta has been divided vertically at its base (J) and the left half taken away. The intra-ventricular septum and the cusps of the aortic valve are exposed. The anterior cusp of the valve is fully exposed and the mouth of the right coronary artery is seen. Directly beneath the posterior end (right hand end in the figure) of this segment, the left division of the auriculo-ventricular bundle enters the ventricle and immediately splits into two chief branches; these two branches lie upon two horizontal bristles, above which there has been a very small amount of dissection. The further subdivision of the branches is perfectly clear, the arborisations are carried in free strands across the cavity; several large branches enter the papillary muscles, the bases of which are seen (P). Two bristles are placed behind finer branches of the coarse network. I lies on the inferior cava; G on the pulmonary artery. Note the large collections of nerve tissue at the base of the heart; bristles are placed behind the thick strands at G, H, and C.

between the muscular trabeculæ by means of bridges completely enwrapped by endocardium, and these bridges are conspicuous at the apex of the left ventricle in almost every heart, be it human or otherwise. It is said that the bundle and its branches are isolated by connective tissue beneath the endocardium until the papillary muscles are reached, and that no union takes place with the ventricular musculature during the earlier parts of the distribution; for no such union has been found up to the present time.

The junctional tissues may be divided for purposes of histological description into the following portions :—

1. The auriculo-nodal junction.
2. The auriculo-ventricular node.
3. The bundle proper.
4. The right and left branches of the bundle.
5. The arborisations and the network of Purkinje.
6. The transitional fibres between network and ventricular substance.

The histological structure of these several divisions varies considerably from one species of animal to another and the most marked differentiation is seen in the hearts of the ungulates. Yet there is much which is held in common. The following description applies particularly to the condition in the dog and in man, in which the resemblance is close.

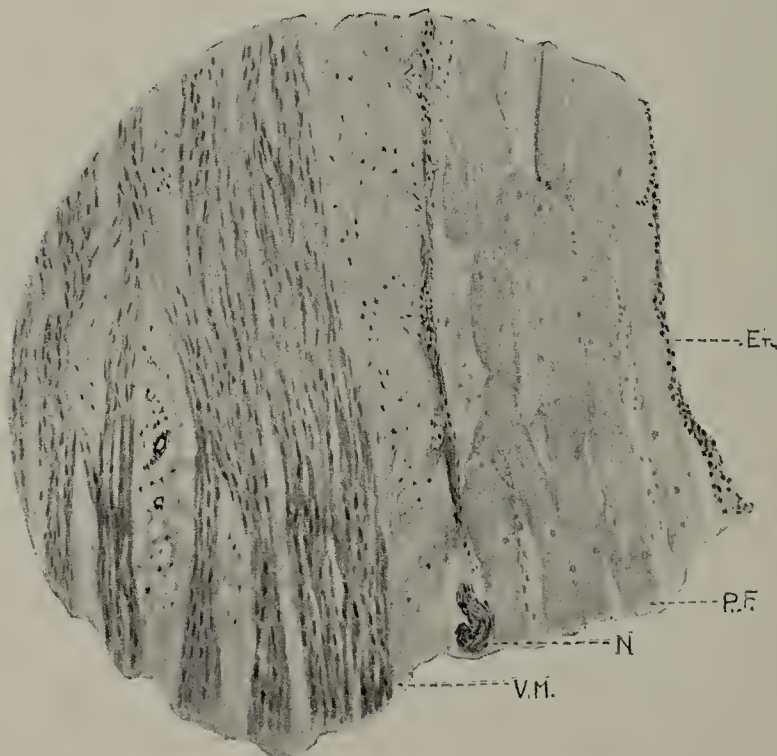


Fig. 4. A longitudinal section of the moderator band of a sheep's heart (magnification 70 diam.). One of the free borders of the band is seen to the right (*En*, endocardium). Directly to the left of the endocardium are a number of longitudinally running and branching Purkinje fibres (*P.F.*). They are separated from the ventricular muscle (*V.M.*) of the band by a loose connective tissue sheath in which a small portion of a nerve (*N.*) is seen. Note the size of the Purkinje fibres and their large pale nuelei.

The auriculo-nodal junction consists of fibres which are smaller than those of the auricle itself and which are arranged mainly in parallel fashion ; the transition to nodal tissue is abrupt, and here the tenuity of the individual fibre is remarkable. The node consists of an intricate interlacement of the slender fibres, which cross and join at all angles. The fibres tend to be of spindle form and they are held apart by a rich network of connective tissue. Nerve fibres and ganglionic cells are scattered profusely amongst them in many hearts.

The fibres of the bundle proper have a more parallel arrangement (they are chiefly parallel in man) and are stouter ; they continue to increase in size as they are traced from bundle to arborisation and network, where they assume the proportions well known and characteristic of the fibres of Purkinje (Fig. 4). They appear swollen, striation is comparatively sparse, the nuclei are large, pale and frequently multiple. According to Tawara, the transition to the ventricular musculature is abrupt and consists in a rapid decrease in size with a corresponding increase in striation.

Thus in the course from auricle to ventricle there is at first a diminution in fibre size (which is extreme at the node), an increase in size (which is extreme in the network) and a subsequent and final decrease. The levels at which the transition to the Purkinje type occurs is very variable in the different species ; but this type is usually well represented in the fibres of the main branches.

The network of Purkinje and the two main branches of the bundle may be readily followed with the naked eye in the freshly opened ventricles of the sheep and calf, and often in the human heart ; a short dissection reveals the bundle itself, and it may be traced with ease to the tissues of the auricle (Fig. 2). The bundle in man and the sheep is from 1-2 mm. in thickness, and its fibres are conspicuous on account of their pallor.

According to the modern view, it is by means of this bundle that the functional union of auricle and ventricle is effected, and it is through this structure that the impulses from the auricle, which initiate the ventricular contraction, are conveyed. They must therefore pass from the neighbourhood of the coronary sinus to the right and left groups of papillary muscles. We shall examine this proposition more closely in a subsequent chapter.

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CHAPTER II.

INTRA-AURICULAR PRESSURE CURVES AND VENOUS RECORDS.

THE fundamental principle of venous pulse work lies in the fact that the change in the volume of the jugular veins is intimately connected with the change of pressure in the right auricle. The venous pulse constitutes an index of intra-auricular pressure. It is consequently of importance that a clear conception should be obtained of our knowledge of intra-auricular pressure as it has been gained from experiments carried out upon the lower animals.

Intra-auricular pressure variations in experiment.

The events of the auricular cycle have received careful investigation by Chauveau,^{1 & 2} Marey,^{2, 20 & 21} Fredericq,^{5 & 6} Frey and Krehl,⁷ Porter²⁵ and others. The experiments have been performed upon the dog and the horse. The curves of Porter were taken with the most delicate recording instruments, attached to sounds introduced into the right auricle. It will be convenient to adopt them as typifying changes in intra-auricular pressure. In common with all other observers Porter found that auricular contraction is accompanied by a positive wave of pressure. This *first positive wave* terminates at or a little before the onset of ventricular systole ; it is followed by a slight dip, *the first negative wave*. The dip would be continued were it not that at the commencement of ventricular systole a *second positive wave* is met with, and this in turn is followed by the *second negative wave*, the most striking fall of pressure in the majority of curves. The depression is continued into ventricular systole in variable degree and gives place, towards the termination of the ventricular contraction, to the *third positive wave*, a wave which terminates at or about the instant when the tricuspid valves open. The relationship of these waves, positive and negative, to other events of the cardiac cycle will be more fully appreciated by reference to the accompanying diagram (Fig. 5).

The venous pulse in animals and man.

Pulsation of the veins of the neck in pathological conditions is often such an obvious phenomenon that it must have been recognised for many centuries. References to venous pulsation can be traced in the writings of Lancisi¹² and Morgagni²² in the early and middle years of the eighteenth century. In 1794, Hunter¹⁰ described pulsation in the veins of a dog.

Later, similar movements were described by Wedemayer³⁰ in the veins of a horse. Friedreich⁸ took venous curves from the neck of pathological subjects in 1865; and two years later Potain²⁶ obtained simultaneous tracings of the apex beat, carotid, radial and jugular pulses from his sister; his description of the events and his interpretation of them are, in the light of our present knowledge, wonderfully accurate. From the time of Potain's contribution, observations have been published by many writers.^{4, 9, 24 & 28} In 1893-4, Mackenzie^{14 & 15} published his first papers, which, with his collected observations¹⁶ on *The Study of the Pulse*, appearing in 1902, have given the impetus to a careful investigation of the whole subject.

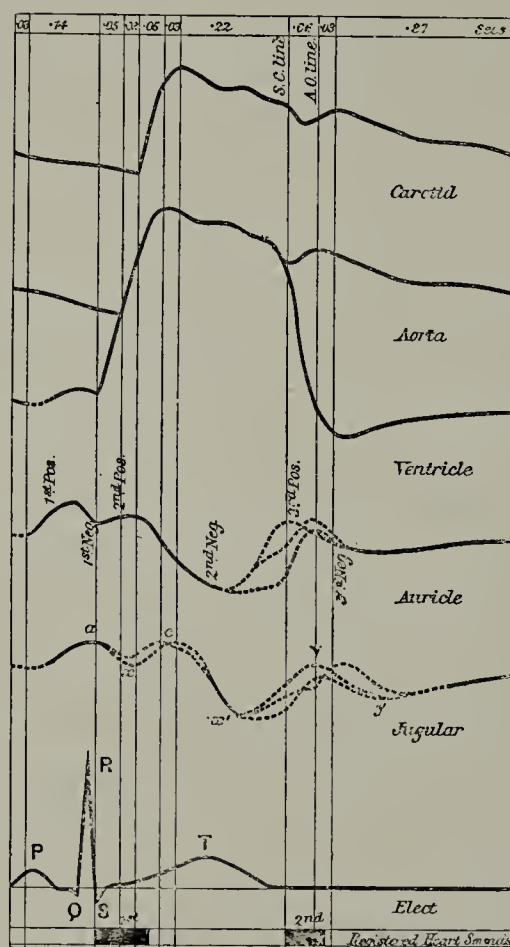


Fig. 5 (republished in modified form from "*Further Advances in Physiology*," Ed. by Hill, 1909). Diagrammatic representation of the pressure changes in the cardiac chambers, and their time relationships to carotid, aortic, jugular, and electrocardiographic curves and heart sounds. The scale of abscissæ is 1 mm. to .02 sec.. The diagram has been constructed from a number of data collected from the chief papers upon the subject. The dotted lines indicate portions of the respective curves which appear to be subject to variation. S.C. = semilunar valve closure. A.O. = opening of auriculo-ventricular valves.

Pulsation in the veins of animals is not confined to the larger vessels feeding the auricles; it is a constant phenomenon in dogs, cats and rabbits, and may be seen extending in many of them into the smaller veins of the

neck and limbs. Gottwalt was of the opinion that the jugular veins of all normal persons pulsate, and this conception is receiving constant confirmation. It is possible at the present time to obtain venous records from the neck of the majority of normal individuals. Pulsation is usually so well marked as to be visible. Any agency which tends to promote a heightened venous pressure, such as raised intra-thoracic or abdominal pressure or gravitation, increases the force and visibility of venous pulsation. It is for this reason that tracings are best taken in the reclining posture, and that in those cases, where pulsation is feeble, expiratory suspension of respiration increases its prominence.

In man, the venous pulse is seen and recorded with the greatest facility in those veins which have but a short distance to travel before reaching the heart. Tracings may be taken direct from the external jugular vein when this is engorged, but more often the receiving instrument must be applied over the *jugular bulb*, as it is termed, which lies a little above and 25 mm. external to the sternal end of the clavicle.¹¹ A needle passed back into the neck at this point strikes the internal jugular vein at a point where it is guarded by a pair of valves, which, when the path to the auricle is obstructed, or when blood is regurgitated, produce a bulging in the vessel from which the jugular bulb derives its name. Passed further on the needle transfixes the subclavian artery.¹¹ Tracings are obtained from the neck, in which the sternomastoid is relaxed, by applying to it, with light pressure, a small receiver, whose width is about 4 cm., and depth 1 cm.. The interior of the shallow cup communicates by air transmission with a delicate tambour carrying a writing style. This simple apparatus is perhaps the most satisfactory as yet invented. The curves obtained from the jugular bulb are frequently complicated by the primary wave of the arterial pulse, which in clinical work is not without its advantages, and by the respiratory movements when these are present. Curves are best obtained from the right side of the neck, for here the course of the innominate is shorter from neck to heart; clinically they may also be taken from the pulsating liver.

With regard to the rest of the mechanism little comment is necessary; when the apparatus described is adjusted to a modified sphygmograph, as in Mackenzie's original instrument, the fitting of a reliable time-marker renders it cumbersome. Another and the most convenient instrument for clinical work is "Mackenzie's ink polygraph" (Fig. 6), with which tracings of any length and at various speeds may be taken.¹⁹ It allows a simultaneous record of any two given pulsations, and carries a reliable time-marker. The curves from the jugular vein give, considering the indirect methods of determining them, surprisingly constant results, and in this lies their chief justification.

In animals, curves have been secured from the vein wall or blood stream by many workers. ^{4, 5, 9, 23, 27 & 28} The results in man and animals are in such general agreement that one description of them will suffice.

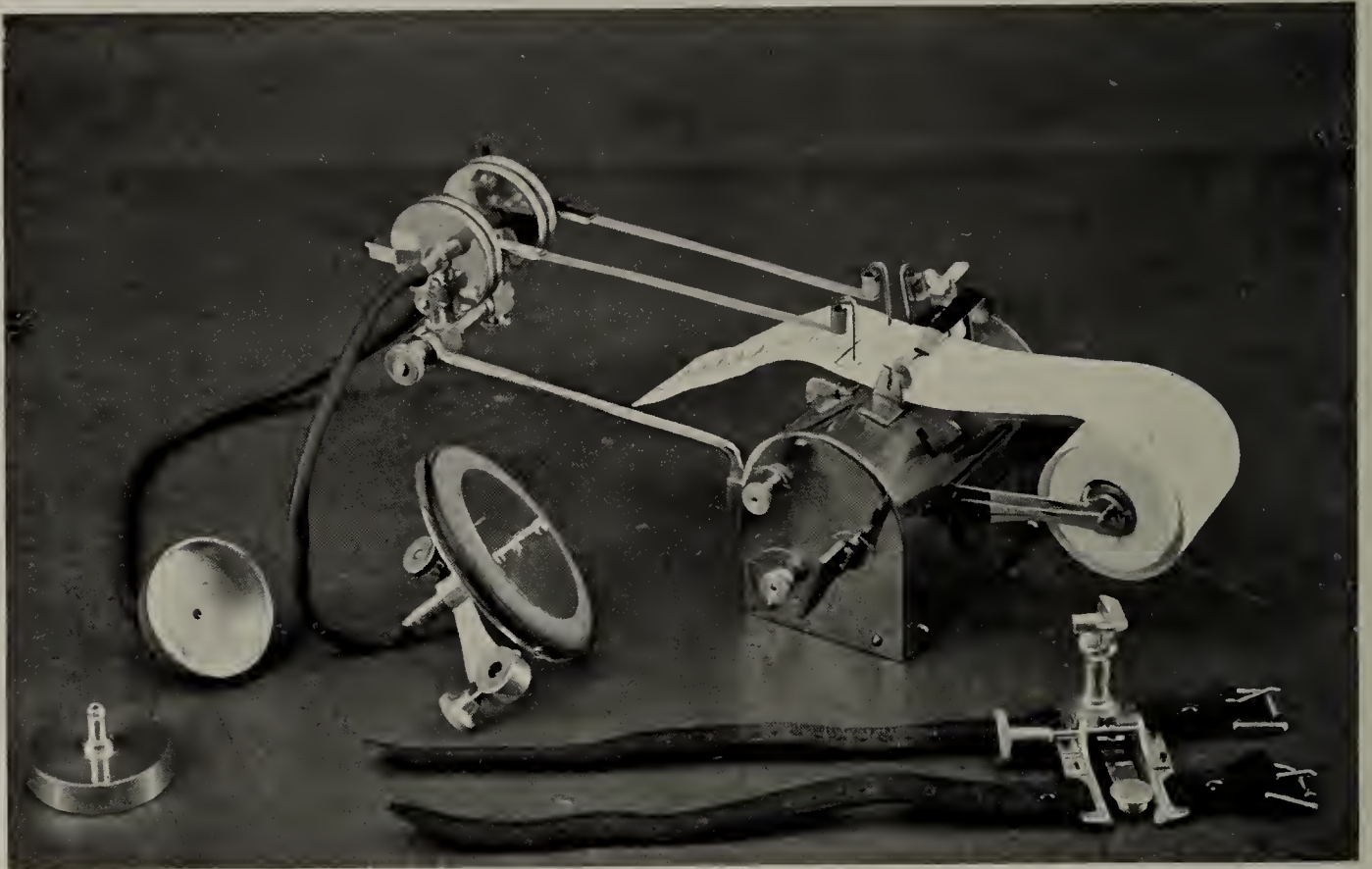


Fig. 6. Mackenzie's Ink Polygraph.*

A picture of the ink polygraph is shown in Fig. 6. It consists of an encased clock to which the remainder of the apparatus is fitted. The reserve paper is held upon a support to the right, and the end of it is carried beneath a roller, which is driven by the clock. It passes directly to the writing platform, and then falls clear. To the left the clock-case is fitted with a stand to which two transmitting tambours are attached in such a way that adjustments in all required directions are accomplished with facility. Each tambour is fitted with a lever and writing style, bearing an ink reservoir (those shown in the figure have recently been replaced by a lighter and improved pattern). The transmitting tambours communicate with the receiving apparatus by means of rubber tubing. The venous or apical receiver is a simple cup; the radial receiver is covered with thick rubber, to which a metal plate and button are attached. When in use, the radial receiver is fastened to the splint (shown in the bottom right hand corner of the figure) in such a way that the button of the receiver lies on a second button in the centre of the splint. This second button is attached to a spring, the bending strain of which can be varied at will. The splint is fastened to the arm by means of leather straps and the button lies over the radial artery. The clock-case contains a separate mechanism driving a time-marker, which records $\cdot 2$ sec.. The clock-case is also fitted with keys for winding the driving gear and time-marker, with an adjustment for regulating the speed and with a lever for starting and stopping the clock.

* As supplied by J. Shaw, Esq., Instrument Maker, Padiham, Lanes.. Price £10 10s., complete.

Speaking broadly, the venous pulse, like the auricular curve, shows three main elevations and three main depressions; the general outlines of venous and auricular curves are such as to leave no reasonable doubt that the factors ultimately concerned in the production of their waves are identical for each, a relation upon which many writers have laid emphasis. The final evidence depends on the detailed analysis of each wave and will be given in subsequent pages. For the time being, it will be convenient to recognise the three main elevations of the venous curve (the *a*, *c* and *v* waves) as corresponding to the three positive waves of the auricular pressure curve, and the three main dips of the venous tracing (the *x*, *x'* and *y* depressions) as corresponding to the three chief negative waves of the auricle.

These events are represented in Fig. 5, and their relations may be tabulated as follows:—

<i>a</i>	<i>First positive wave.</i>
<i>x</i>	<i>First negative wave.</i>
<i>c</i>	<i>Second positive wave.</i>
<i>x'</i>	<i>Second negative wave.</i>
<i>v</i>	<i>Third positive wave.</i>
<i>y</i>	<i>Third negative wave.</i>

The majority of writers are agreed in timing the commencement of the *c* wave in man and animals as synchronous or almost synchronous with the primary wave of the carotid at the same level of the neck.

The *a* wave and *x* depression have together a duration of .1 to .2 sec. and constitute the *a-c* interval. This *a-c* interval, the time distance between the beginning of the waves representing the commencement of auricular and ventricular systole, is taken as a measure of the function of the heart in respect of the conduction of impulses from auricle to ventricle, and is of great clinical importance.

The relationships of *v* are variable; it may commence during the systolic plateau or towards its termination, and the changing position of its onset probably depends largely upon the state of engorgement of the right heart. Its termination in the *y* depression is almost universally stated to be synchronous with the opening of the auriculo-ventricular valves.

The cardiac events upon which the waves of the auricular pressure and jugular pulse curves depend.

The first positive or "a" wave.—The presystolic onset of this wave leaves little doubt that it is due to the contraction of the auricle and it is universally attributed to this cause. It disappears from the tracing, when as a result of tetanisation of the auricle this chamber ceases to beat.⁵ In cases of partial heart-block in which the auricles maintain a rhythm which

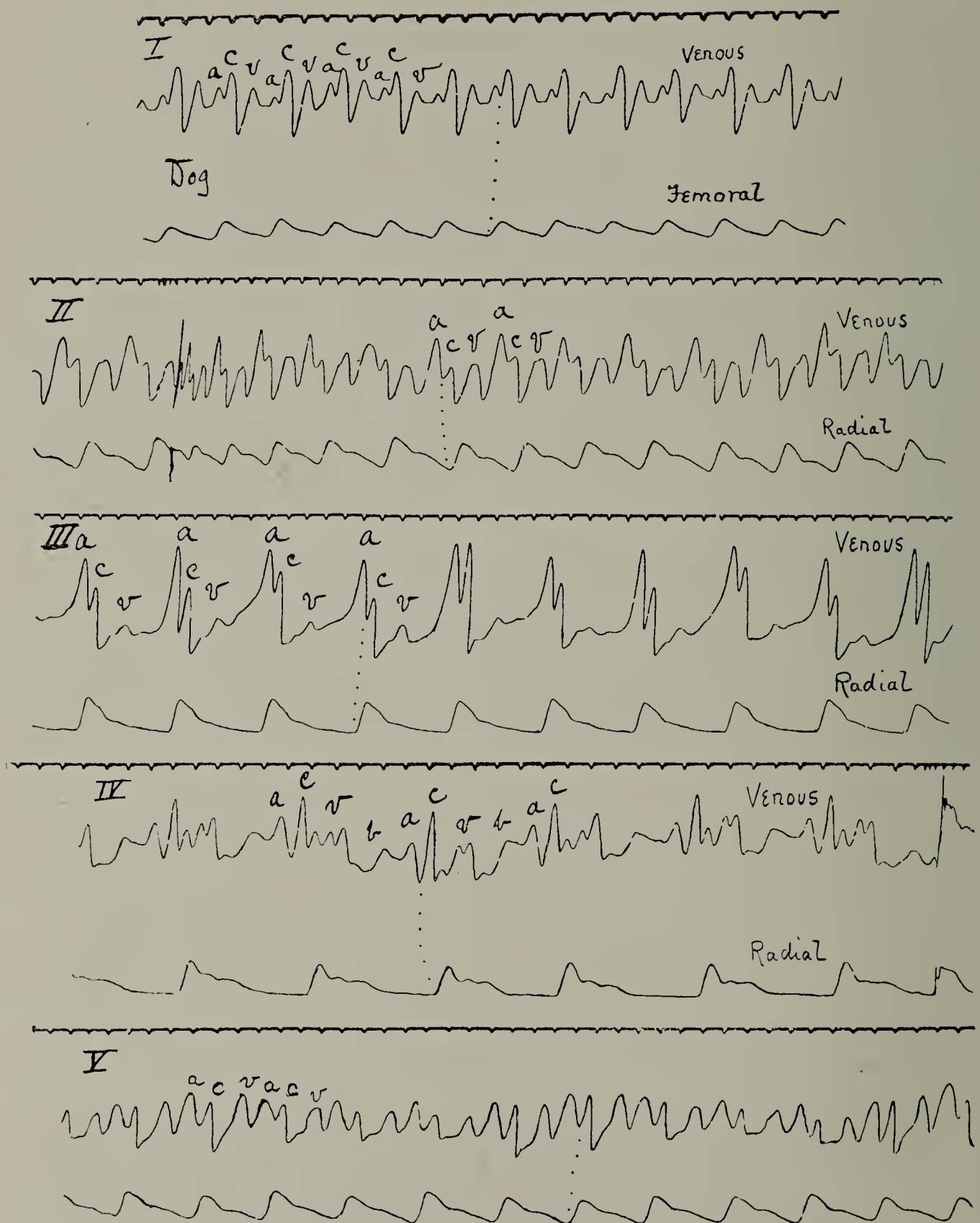


Fig. 7. Physiological forms of the venous pulse.

Polygraphic records.—I, simultaneous femoral and jugular curves taken from a dog. II-V, simultaneous radial and jugular curves from human subjects; *a*, *c*, and *v* waves are clearly shown in each curve. In IV, *v* is split, and several additional waves, including *b*, are present. Note the constancy of form from cycle to cycle in each instance.

is a multiple of the ventricular rhythm, the wave occurs more frequently upon the jugular tracing and at equal time intervals. Experimentally it has also been shown that cessation of ventricular contraction does not affect it.⁵ The wave is consequently established as due to auricular contraction and auricular contraction alone.

The wave *a*, as it occurs in the neck, is probably in the main a volume effect; for with the cessation of flow into the heart the veins act as a reservoir. It is possible, nay probable, that in some instances there may be in addition a certain grade of regurgitation from the auricle, or that a centrifugal pressure wave resulting from auricular contraction aids in its production.*

The second positive or "c" wave.—The dependence of the wave *c* upon ventricular systole has been clearly established experimentally⁷ and clinically, but the actual factors involved in its production have given rise to considerable discussion. From the collected evidence we may conclude that the second positive wave is a real event in the auricle of man,³ and that it may appear in the neck as a component part of the jugular pulse. There can be no doubt that in many tracings of the jugular pulse the shock from an artery lying in the neighbourhood of the receiver also aids in its production.⁸ Whatever the ultimate factors involved, and many have been ascribed, the practical conclusion is unaffected. The *c* wave in the jugular may be safely taken for clinical purposes as synchronous with the primary wave of the arterial pulse at the same level of the neck, and it forms a valuable standard in the interpretation of tracings.

The third positive or "v" wave.—The third positive wave in the auricle is assigned to several causes. Its dependence upon a ventricular rather than upon an auricular event has been clearly established experimentally and clinically. Unquestionably the damming back of blood and rise of pressure in the auricle, resulting from ventricular systole, is a factor of great importance. It has been associated also with the sudden release of the base of the ventricle at the commencement of ventricular diastole. It is probable that both factors play a part in the production of the wave under certain circumstances; that with a quick filling of the auricle or sustained plateau the first will be prominent, and that with a slower filling or quicker heart beat the pressure in the auricle will be low when the ventricle passes into diastole, and that as a consequence the tricuspid valves will open somewhat later. Under these circumstances the second factor may be more pronounced. If the view is accepted that both are contributory causes, the occasional division of the wave into two component parts (Fig. 7 *IV*) becomes more intelligible.

* Those who require a more detailed account of the physiological venous pulse are referred to an article in "Further Advances of Physiology," edited by Leonard Hill, London, 1909.

The first and second negative waves or "x" and "x'" depressions.—These two negative waves, which together form the most prominent depression of the auricular and venous curves, are probably due to three causes: the enhancement of the negative pressure in the chest, consequent upon ventricular systole; the auricular relaxation dependent upon the originally low intra-thoracic pressure; and the dragging down of the auriculo-ventricular ring as a result of ventricular systole. Of these causes, the first is insignificant in its effects, the second is most active in the early phases, and the third most prominent in the later phases of the depression.

The third negative wave or "y" depression.—With the diastole of the ventricle, the pressure within it falls rapidly and may become markedly negative. The fall of the pressure is accompanied by the opening of the auriculo-ventricular valves, and the blood contained in the auricle passes into the ventricle. As a consequence the pressure in the auricle falls, and the depression in the auricular tracing timed to occur with the opening of the valves is universally attributed to this cause.

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CHAPTER III.

THE GALVANOMETRIC METHOD AND PHYSIOLOGICAL ELECTROCARDIOGRAM.

The string galvanometer.

GALVANOMETRIC instruments are based upon the principle of the interaction of a magnet and a conductor of current. In the familiar Kelvin galvanometer of the physiological laboratory, a small magnet to which a mirror is attached is suspended by a fine thread. The magnet is surrounded by coils of wire, and with the passage of currents through the latter the magnet is deflected, and a beam of light reflected from the mirror serves as an index of such movement.

The string galvanometer, in its present form the invention of Einthoven,⁴ is built on the opposite principle. A single conducting strand lies between the two poles of a powerful magnet. Currents passed through the string induce deflections of it. The sensitivity of the instrument and the quickness of the movements have been increased by decreasing the weight of the string and by augmenting the strength of the magnetic field in which it lies. The poles of the magnet are closely approximated, so that a narrow chink separates them. It is in this cleft that the string is fixed, and its movements are observed by projecting its shadow upon a screen. The strings employed are extremely delicate, consisting of finely drawn platinum or of a film of silver over a finely drawn quartz thread. In thickness the fibre is .002 to .003 mm., and it is attached to a carrier at the upper and lower ends of the cleft between the magnetic poles. Through the carrier the terminations of the filament are connected to the two wires which convey the current to be investigated to and from the instrument.

A full account of the history of electrocardiographic studies will be found in the papers to which reference is made at the end of this chapter. The main steps may be given quite briefly.

In 1856, Kölliker and Müller⁹ first demonstrated the presence of a current of action in the heart; and they were able, by laying a frog nerve-muscle preparation in contact with a beating heart, to show the presence of two distinct electrical changes at each beat of the ventricle. Their observations were followed by those of a number of investigators^{8 & 25} working with the earlier types of galvanometer. At a later period the capillary electrometer was used, and, employing this instrument, Waller²⁷ (in 1889) first showed the possibility of registering the human heart beat.

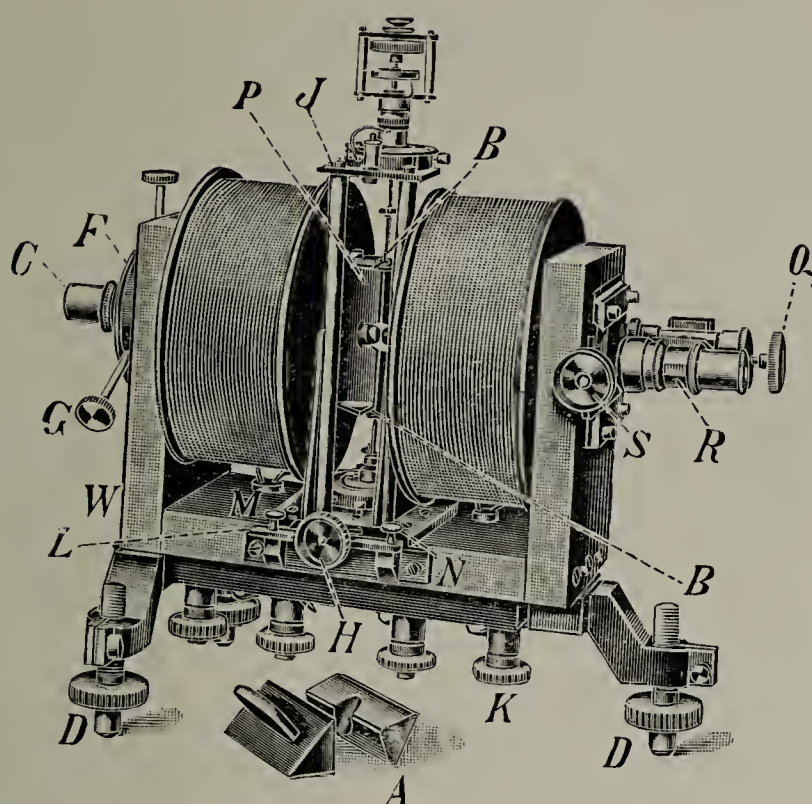


Fig. 8. Einthoven's string galvanometer* (large pattern), as supplied by Dr. Th. Edelmann and Son, of Munich.

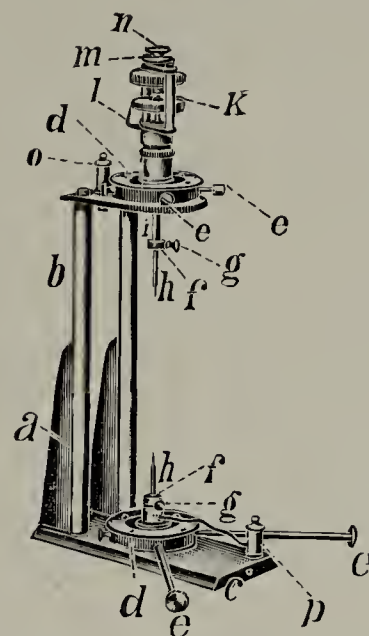


Fig. 9. The string carrier, which slides into the centre of the galvanometer.

The string galvanometer in its complete form, and as supplied by Messrs. Th. Edelmann and Son of Munich, is shown in the accompanying figure (Fig. 8). It stands on four adjustable feet (*DD*), to which the base (*W*) is attached. The base supports two heavy iron blocks surrounded by coils of wire (the total resistance of which is approximately 9 Ohms), which are joined in series and are connected to accumulators (20 Volts) by means of the terminal screws (*K*) on the undersurface of the base. The poles (*P*) of the magnet are bevelled, and are closely proximated at the centre of the instrument, so that a narrow and vertical cleft is left between them (*BB*). The projection microscope (*R*) (Apochromatic 16 mm. focus) lies to the right, the condensor (*C*) (Achromatic AA) to the left; they are driven through the centres of the poles, so that the lenses lie one on each side of the slit (they are seen in Fig. 8). The condensor is centred by means of screws (*G G*). The projecting microscope is focussed by means of a screw (*Q*), and is adjusted in the lateral direction by means of a screw (*S*). The string is carried in a special holder (*J*), and this slides into the instrument in the grooves (*N*). The depth to which it is buried in the instrument is adjusted by means of a screw (*H*) working against a spring on the opposite side. With the string in place the instrument is closed by means of triangular blocks of brass (*A*), and small sliding glass caps at top and bottom (which are not shown in the figure). The string carrier is illustrated in Fig. 9. It consists of a stand (*a*), a base (*c*), and a roof (*d*). The roof and floor support the pointers (*h*) to which the string is attached. The pointers are bound in place by clamps (*g*) and they are centred by means of four screws (*e, e*). The tension of the string is adjusted by the apparatus *n, m, k*: the adjustment is fine and coarse; the fine adjustment is provided with a scale and indicator (*l*). At *o* and *p* are two terminals with which the string is in direct connection, and to which the wires carrying the *tested* current are attached. In using the instrument the image of the wire is projected by means of an arc light on to the horizontal slit of a camera. Behind the slit a photographic film moves in a vertical direction. The movements of the shadow are in a horizontal direction, that is to say at right angles to the slit.

The patient or animal is connected to the instrument by means of a system of wires and electrodes, a simplified and diagrammatic scheme of which is shown in the accompanying figure (Fig. 10).

* A complete installation, sufficient for most clinical purposes, can be fitted up for £120,

ERRATUM.

Page 21, *ten lines from the bottom of the page*
"right leg" should read "left leg."

K^6 is opened and the string returns to its original position. The instrument is of such sensitivity that with a magnification of 400-600 diameters and the patient in circuit $1/1000$ of a Volt yields a deflection of approximately 10 mm.. When the current from the accumulator is thrown into the circuit, the total deflection of the string occupies an appreciable time; measured it should amount to approximately .02-.03 sec.. Under these conditions²³ the movements of the string accurately represent the current changes which accompany the heart beat.

The patient is thrown into circuit by opening K^2 . At the time K^3 should be closed and the string relatively tense. A permanent deflection of the string usually results, as a result of skin activity. It is compensated by throwing in a small current from the compensator in the reverse direction. With K^6 and K^5 closed an E.M.F. of $1/20$ Volt exists between the ends of the wire (WW). Fractions of this are suitable for employment in compensating. When compensation is secured, K^3 may be opened, and electrocardiographic curves obtained with the string reduced to an appropriate degree of slackness. The less tense the string the greater is its excursion with a given current, but a certain degree of slackness should not be surpassed or the curves suffer serious modification in outline. The limit of safe slackness is estimated by measuring the deflection time of the string in the manner previously recounted (Fig. 11).

For further details of technique the reader is referred to the publications of Einthoven,⁵ Samojloff^{23 & 24} and others.¹⁴

The first satisfactory curves from the mammalian heart were obtained by Bayliss and Starling.²

In 1903 Einthoven introduced his new instrument, the string galvanometer. The ease with which records are obtained by the utilisation of this instrument and the truthfulness of the curves obtained make it probable that it will supersede the majority of instruments of the same class. It is an instrument which may be used as a routine method of recording the heart beats in experimental researches, and its introduction has brought the systematic electrical examination of patients within the field of practical medicine.

The physiological electrocardiogram.

The leads adopted.—Electrocardiographic curves may be obtained by leading off from various points of the body; but except for special purposes two contacts are generally employed, one of which lies towards the base, the other towards the apex of the heart. For the examination of clinical subjects Einthoven^{5 & 6} adopts three leads, taking, in pairs, the right and left arm, the right arm and left leg, and finally the left arm and right leg; and these have come into general use.

It should be known that the type of electric curve which is obtained from the heart depends upon the lead chosen, and that no two leads give precisely the same picture (Fig. 11). Each separate lead is of considerable service, for one may give information which is not conveyed by another. All show indications of the contraction of both auricle and ventricle. As it is the chief purpose of this book to deal with the disordered mechanism of the heart beat, we shall confine ourselves in the main to a description of the curves obtained by a single lead, namely the right arm and left leg. And this lead is chosen because, as a matter of experience, it is found to

be the most serviceable, and because it probably most clearly represents leads obtained directly from the base and apex of the heart in animal experiment. Except where specifically stated to the contrary, it should be understood that the electrocardiographic figures which illustrate the succeeding pages were obtained by a lead of this kind, and the statement applies to both clinical and experimental curves.

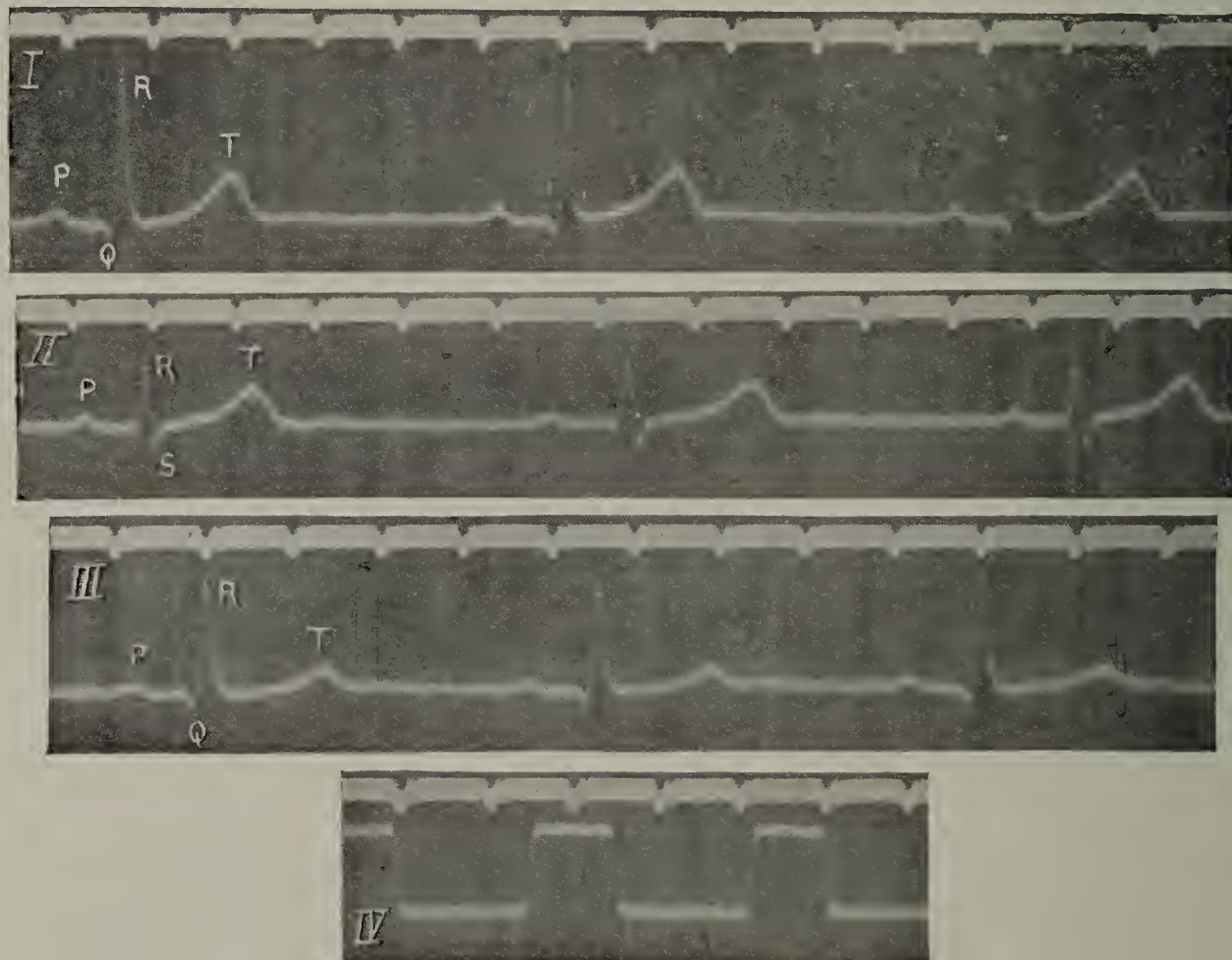


Fig. 11 (\times approx. $\frac{5}{6}$). Three electrocardiograms (I-III) from a young and healthy adult, and a control curve (IV).

- I. Leading off from the right arm and left leg.
- II. Leading off from the right arm and left arm.
- III. Leading off from the left arm and left leg.
- IV. Deviations obtained by throwing in and cutting out an electromotive force of $\frac{1}{1000}$ Volt (string resistance 9500 Ohms), with the string at the same tension. In the original figure the movements were magnified 410 times. The deviation time is .02 sec..

Note the amplitude of the several variations in the separate leads, and the appearance of *Q* in I and III and of *S* in II. *P* represents auricular, *Q*, *R*, *S* and *T* represent ventricular contractions. The time-marker, in this and all succeeding figures, beats .2 sec., except where definitely stated to the contrary.

The physiological type of mammalian electrocardiogram.—It is difficult to define the exact limits of the physiological electrocardiogram, for no two individuals present exactly similar curves.⁵ But, continuing to speak of the lead from right arm and left leg, there are certain features which are common to electrocardiograms as a whole. The electrocardiogram of the normal subject consists of two parts, an *auricular* and a *ventricular complex*.

The *auricular complex* consists of a primary deviation in the base-negative* (or upward) direction. Adopting the terminology of Einthoven, it is termed the summit or peak *P* (Fig. 11). This summit, which is either rounded or pointed, is succeeded by a horizontal line (an isoelectric† period) or by a less prominent deviation in the opposite (base-positive or downward) direction.

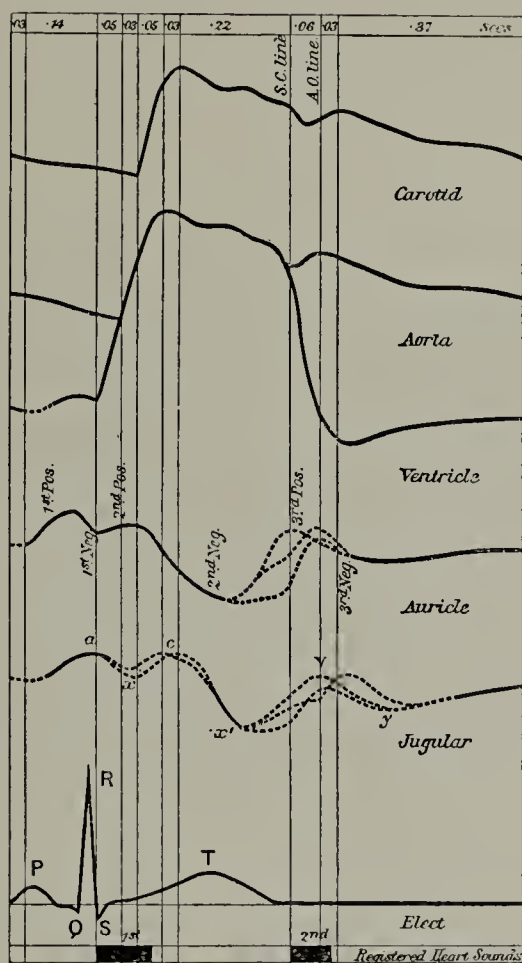


Fig. 12.

The *ventricular complex* is, as a rule, triphasic, and is constituted by the variations *R*, *S* and *T*, of which *R* and *T* are directed upwards, while

* A term which is explained later.

† The contacts with the patient are isoelectric when no current is passing through the string.

S is directed downwards. *R* is usually the most conspicuous summit in the curve, its duration is short (usually $\cdot 03$ sec. or less). It may be preceded by a small and brief deviation in the opposite direction (the summit *Q* of Einthoven); it is followed by a brief downwardly directed deviation *S*, which is of very variable amplitude. As a general rule the latter is inconspicuous; it may be prominent.

The opening phases of the electrocardiogram consist, therefore, of a summit *P*, associated as we shall see with auricular contraction, and summits and dip *Q*, *R* and *S* associated with the initial events of the ventricular systole. This group of variations is followed by a longer or shorter line which is horizontal, during which the contacts are isoelectric, and the whole curve ends in a broad and prolonged variation *T*.

The chief time relationships of the electrocardiogram.—Numerous electrocardiograms have been taken simultaneously with records from the auricular and ventricular musculature, with intraventricular pressure curves,^{14 & 15} with the heart sounds¹⁷ and with polygraphic curves.^{3 etc.} The relationships of the several events are portrayed in the diagram (Fig. 12). The measurements of the writer are in general agreement with those already

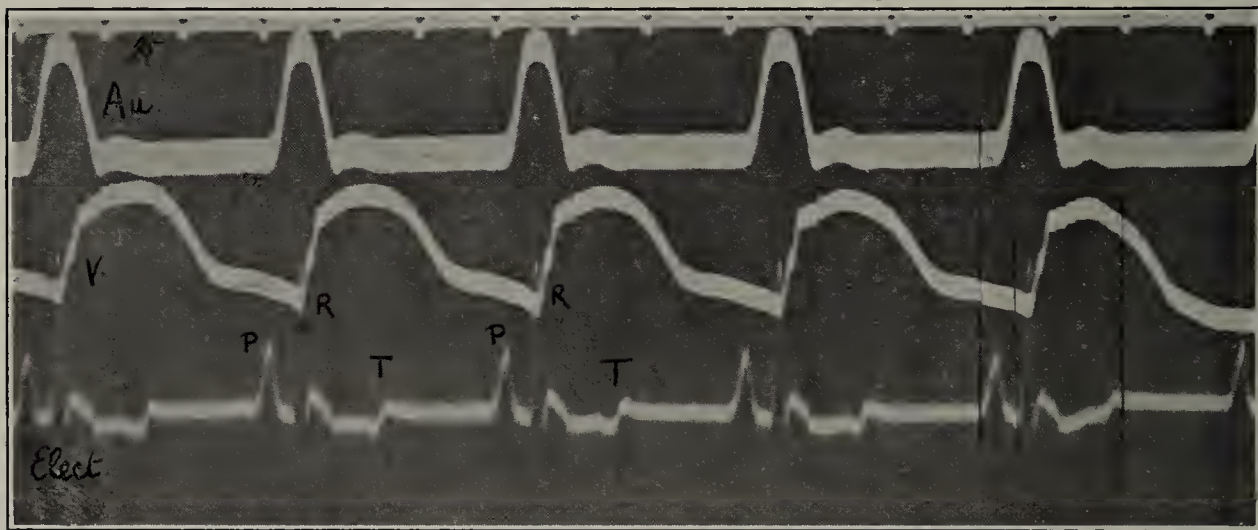


Fig. 13 ($\times \frac{5}{2}$). Simultaneous myocardiograms (*A*=auricle, *V*=ventricle) and electrocardiogram from a dog. Showing the time relationships of the several events in the three curves. The auricular upstroke is $\cdot 04$ sec. later than the upstroke of *P*. The ventricular upstroke is $\cdot 03$ sec. later than *R*. *T*, which is partially inverted, ends during the ventricular systole. The myocardiograms of this and subsequent figures have little or no transmission delay; they were taken with a modification of Roy and Adami's instrument; the pull was direct from heart muscle to lever.

published. *P* stands in relationship to auricular systole and its upstroke precedes *R* by $\cdot 12$ – $\cdot 17$ sec. (the *P*-*R* interval) in normal human subjects. The upstroke of *R* usually precedes the onset of ventricular contraction (as estimated from myocardiograms from the front of the ventricle, intraventricular pressure curves and heart sound registration) by approximately $\cdot 03$ sec. (Fig. 12 & 13). The interval has been regarded as a measure of the

latency of the contraction. But it has been shown by Kahn,¹⁶ and the writer is in agreement with him, that when the muscle is artificially excited in the neighbourhood of the attached myocardiograph lever, the interval between the appearance of a string deviation and the first detected shortening of the muscle is much less (according to Kahn it amounts to .002 sec., or even less). In all probability the interval .03 sec. chiefly represents the time between the commencement of contraction of the deeper layers of the musculature and the appearance of contraction in the main mass, the middle and superficial layers of the walls.

The summit *T* falls during the systole of the ventricle, when the whole mass of the muscle is shortened. It subsides a few hundredths of a second before the end of the plateau, and immediately (.03 sec.) before the occurrence of the second or aortic sound.¹⁷

Thus the events *R*, *S* and *T* are attributable to ventricular systole. *P* lies in presystole, and is contemporaneous with auricular contraction.

When dissociation of the auricular and ventricular rhythms is present the summit *P* is found at uniform intervals in the curve, but bears no constant relationship to the ventricular complexes which are likewise present. Under these conditions the auricular complex sometimes shows a second phase directed downwards, and the whole complex is recognised as diphasic. That *Q* belongs to ventricular and not to auricular contraction is shown by the fact that in cases where it is present, it immediately precedes *R*, whether the auricular complex occupies a presystolic position or not (Einthoven's curves⁵).

That *P* is the representative of auricular activity and the remaining deflections represent ventricular activity is also clearly demonstrated by special leads from the exposed heart.^{11 & 23} Thus, while a lead from the entering systemic veins and the heart apex shows *P*, *R*, *S* and *T* variations, a lead from the auriculo-ventricular groove and apex yields *R*, *S* and *T* only.

The meaning of the electrocardiogram.—The galvanometer and registering apparatus is so arranged that when a current is passed from a copper-zinc couple and from above downwards through the string, the photographic film eventually shows a deviation of the shadow to the left; a deviation in this direction is upward in the figures which illustrate this book. When the leg electrode is connected with the upper end of the string (Fig. 10) and the arm to the lower end, the three chief summits *P*, *R* and *T* of the electrocardiogram point in the upward direction in the figures. Now a lead from the right arm represents a lead from the tissues abutting upon the base of the heart, while a lead from the left leg represents a lead from tissues surrounding its apex. When, as a result of the heart beat, a deviation of the string is recorded in the upward direction, it is customary to state that the arm electrode and the tissues with which it is connected have become *negative* relative to the leg electrode and the tissues in contact

with it. If the arm electrode is connected to the bottom of the string, or if the zinc terminal of a Daniel cell is connected to the same point, R and the deflection caused by the current from the battery are in the same direction. The heart during the initial phase of its contraction may be regarded as a battery, in which the basal portions of the ventricle correspond to the zinc terminal of a Daniel cell. What is the meaning of the direction of this deflection R ? It is an indication of *relative negativity*, and, as we shall see, of *relative activity* of the base of the ventricle.

When the ends of a strip of inactive muscle are connected to a galvanometer, and one end of the muscle is excited, this end manifests a similar electric disturbance, it becomes negative relative to the inactive end.

But if a contraction is initiated in a strip of muscle, the contraction spreads as a wave and travels throughout the length of the tissue. Consequently when one end of the muscle is stimulated, the excited end first shows relative negativity, while as the contraction subsides at this the proximal end, and culminates at the distal end, the latter in virtue of the activity developed in it becomes in turn relatively negative. Leading from the two ends of the strip the galvanometer shows two deviations, of which the directions are opposite. The complete phenomenon is termed a *diphasic effect*.

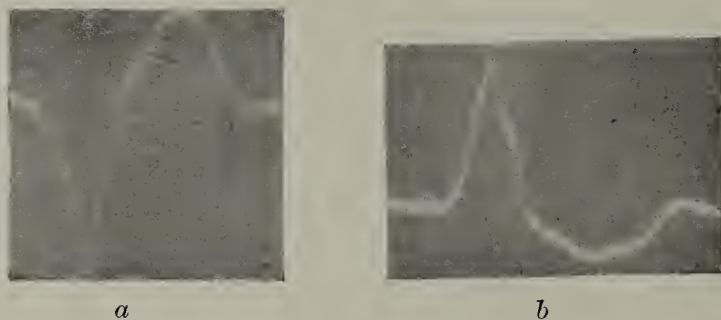


Fig. 14. Two ventricular complexes obtained by stimulation of the apex of the heart (to the left in the figure) and the base of the right ventricle in front (to the right in the figure). The curve excited from the apex is diphasic, the first variation is in the apex-negative (downward) direction, the second variation is in the base-negative (upward) direction. The curve excited from the right base shows the reverse picture, primary base-negativity, and secondary apex-negativity. Taken from a dog.

The interpretation of the electrocardiogram and the determination of the course taken by the contraction wave in the heart muscle which produces it are part and parcel of one question. If electrodes are attached to the basal and apical regions of the quiescent heart and contractions are excited first in the basal and secondly in the apical portions of the tissue, they give rise to curves which are the reverse of each other. The apical excitation (Fig. 14a) yields an approximately diphasic curve, of which the first deviation is downward (apical negativity or apical activity) and of which

the second deviation is upward (basal negativity or basal activity). On the other hand, basal excitation gives rise to a primary deviation in the upward and a secondary deviation in the downward direction (Fig. 14*b*). Briefly, it has been shown that in virtue of its activity the excited point becomes *negative relative* to all other points of the musculature, and that the wave of negativity travels, as does the contraction wave, in all directions, from the point of excitation.^{8, 20 & 25}

The physiological type of ventricular electrocardiogram is a complex curve; it consists of three or four deviations (*R*, *S* and *T* or *Q*, *R*, *S* and *T* respectively) from a basal or isoelectric line. It is consequently triphasic or quadriphasic. The difficulty of interpreting the events upon which it depends are very great, and at the present time it cannot be said that we have more than a superficial grasp of the factors which determine its component parts. The difficulties consist, firstly in the complexity of the paths by which the contraction wave enters the ventricle, and secondly in the extreme variation in the course of the fibres composing the ventricular wall.

The anatomical evidence for the passage of the auricular impulse along the divisions of the bundle and into the arborisations in the neighbourhood of the papillary muscles of right and left ventricles has been given, and we shall presently consider the physiological evidence, which fully confirms this view. The contraction of the ventricle is assumed as starting at a number of points and as spreading from these points. The mass of ventricular substance consists of three main layers, an outer, of which the fibres are arranged in spiral though mainly longitudinal fashion, a thick middle layer in which the fibres are disposed mainly in circular manner, and an inner longitudinal system, to which the papillary muscles belong. Connecting bridges have been described between the last named and the first two.¹

In view of these facts and the early stage of the investigations, no very definite idea of the direction of the contraction wave or of the constitution of the physiological electric curve can be entertained. A great many theories have been put forward, but they are all of a tentative nature. The writer does not propose to comment upon these explanations in any detail, but refers the reader to the special papers on the subject. At the same time it may be well if a general working hypothesis is presented, for it may be of help in emphasising certain facts and certain difficulties. But it should be clearly understood that the suggestions are almost purely hypothetical and that they are not expressed as adopted views.

It has been found that the right ventricle contracts somewhat earlier (·02 sec.) than the left,²⁶ and special investigations suggest that the first point at which the ventricular muscle becomes active is represented by the papillary muscles,^{12 & 22} especially those of the right ventricle. The deviation *R* is consequently considered as the result of contraction of these muscle.²¹ Excitation of the large papillary muscles of the right

ventricle by means of an internal electrode gives rise to a curve of which the first deviation is in the upward direction (Fig. 129 *I* and 130 *IV*). But it is not a duplicate of the physiological summit *R* in the same animal, and this may be due to the more widespread influence of the impulse through the arborisations to neighbouring points under normal conditions. If the ventricular electrocardiogram commences in a peak *R* it may be held to represent contraction of the region in the neighbourhood of the right group of papillary muscles, for they lie towards the base of the ventricular muscle as a whole. If, on the other hand, the cardiogram opens with a *Q* deviation, it is probable that the impulse has been first distributed to parts of the muscle which lie more in the vicinity of the apex (Einthoven). The spread of the contraction wave to the general mass of muscle follows and other contraction waves are initiated in the left side. These are the opening events of the ventricular electrocardiogram, and according as the bulk of the tissues at the apex enters contraction, and its electric effects are unopposed by those of the contraction which is spreading towards the base, the deviation *S* will be more or less marked. A period follows during which the whole ventricle is contracting and during which base and apical effects tend to counteract each other; this period is represented by the horizontal portion of the complex. The deviation *T* succeeds this almost isoelectric portion of the curve and is explained as due to dominating activity of the base of the heart. Gotch^{10 & 11} has brought forward evidence to show that it is specially associated with the duration of contraction in the musculature surrounding the outlet of the ventricle, the muscle abutting upon the base of the aorta (and pulmonary artery). If we adopt the view expressed by this writer, we may regard the general course of the contraction in the ventricle as following the original arrangement of the embryonic heart tube. The contraction proceeds at first in a general direction from base (inlet) to apex (base active, *R*; apex active, *S*) and finally returns and predominates at the base (outlet) (base active, *T*).

Before proceeding to the subsequent chapters, it may be well to emphasise once more the tentative character of this description and to point out that, while we are by no means conversant with the actual course of events, we are certain of the essential facts. The peak *P* is an accompaniment of auricular, the summits *Q*, *R*, *S* and *T* of ventricular contraction. In a given lead, the auricular complex *P* and the ventricular complexes *Q*, *R*, *S* and *T* are absolutely dependent upon the direction of contraction in the chambers in which they are produced; and deviations from the physiological type from beat to beat must be interpreted as the result of variations in the course of the contraction waves.

These are the outstanding facts upon which the interpretation of disordered cardiac mechanism is founded.

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CHAPTER IV.

THE PACE-MAKER OF THE MAMMALIAN HEART.

IN the hearts of the cold-blooded vertebrates the great veins, systemic and pulmonary, terminate in the first chamber, the sinus venosus or *saccus reunians* of His, and there is a clear line of demarcation between the sinus and the auricle proper into which it leads. Consequently it is a matter of little difficulty to show that the heart beat arises as high up as the line of union in these animals. A ligature or clamp placed upon the sino-auricular junction (first Stannius ligature), and thereby isolating the sinus, brings the lower chambers to a condition of standstill, while the upper portion of the dissociated musculature preserves its original rhythm. In the mammalian heart the sinus and auricle are more intimately connected and there is no defined boundary between them. It is natural to seek the remains of sinus tissue at those points at which the great veins enter the heart, and for many years the *sulcus terminalis*, a groove on the outer surface of the auricle, has been regarded as the line of separation of the representatives of the two chambers. Recent anatomical research, for which we are chiefly indebted to Keith, has led to a revision of our ideas upon this subject. As described in a previous chapter, Keith holds that there are sinus remnants in the region of the mouth of the superior vena cava, in the coronary sinus, in relationship to and in the auricular septum, and possibly also at the mouths of the pulmonary veins.

The anatomical and morphological evidence leads us to a belief that the pace-maker lies in one or other of the primitive rests, and directs attention more particularly to the specialised sino-auricular node at the junction of the superior vena cava and the right auricle; for here there is a relatively large collection of specialised muscular tissue, which stands in intimate relationship to the rich supply of nerves entering in this neighbourhood.

Many experimental methods have been adopted in the search for evidence of the position of the pace-maker in the mammalian heart. A number of them are summarised at the end of this chapter. The majority have dealt either with hearts subjected to considerable and direct mechanical or chemical injury, or with hearts in which normal nutrition was disturbed.

The electrical methods outlined in the succeeding pages are free from these objections. Previous workers have also contented themselves for

the most part with observations which appear to locate the pace-maker somewhere in the neighbourhood of the mouths of the great systemic veins.*

It is obvious that, if we can obtain indications of the direction in which the auricular tissue contracts, we shall be in possession of an important clue to the point at which the contraction starts. While the auricular systole may be identified by mechanical means, it has been found impracticable to analyse the direction of the contraction wave in the undamaged heart by this method. Now electrocardiographic curves are of peculiar service in this respect, and we may therefore turn to the facts derived from the utilisation of the galvanometric records.

The electric changes accompanying the auricular contractions, normal and excited.

In the first instance, it is known that the electric curve yielded by an unexposed and naturally beating auricle exhibits very great constancy from animal to animal and from one species of animal to the next. So we may conclude that wherever the pace-maker lies it has a very similar location in different animals of the same species, and in animals of separate species (man, dog, cat and rabbit).

Again, auricular contractions may be excited from any desired part of the auricular musculature by means of single induction shocks, and such experiments may be performed under extremely favourable conditions, with natural respiration, with the thorax closed and with the heart beating at a normal rate.¹⁶ Excitation of many points of the auricular musculature (right and left) immediately reveals the fact that the auricular electric complex obtained when a heart beat is excited from the neighbourhood of the superior vena cava resembles the normal auricular complex very precisely. The interpretation of this observation is clear when we remember

Fig. 15 ($\times \frac{1}{2}$) (*Heart*, 1910-11 II, 23, *Fig. 4*). Three electrocardiograms taken from a dog. In each of the three strips the termination of a tachycardia excited from a fixed point of the auricular tissue is shown at the second or third beat of the strip. The last beats in each strip represent the return of the heart to the normal mechanism as a result of cessation of stimulation (the signal in the top line of each strip fixes the points of stimulation). Attention is directed to the shape of the auricular complex (*P*) in the opening beats of the three strips. They represent auricular contractions propagated from the superior vena cava (*I*), the mouth of the coronary sinus (*II*), and the left appendix (*III*). The *P* waves to right and left in the first strip are duplicates. In the succeeding strips the excited beats are anomalous in outline.

Fig. 16 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 23, *Fig. 6*). Similar curves from another animal. The sites of stimulation were the superior vena cava (*I*), the mouth of the coronary sinus (*II*), and the pulmonary vein area (*III*).

* With the exceptions of Flack's recent observations, the galvanometric examinations have alone been directed to a nicer localisation.

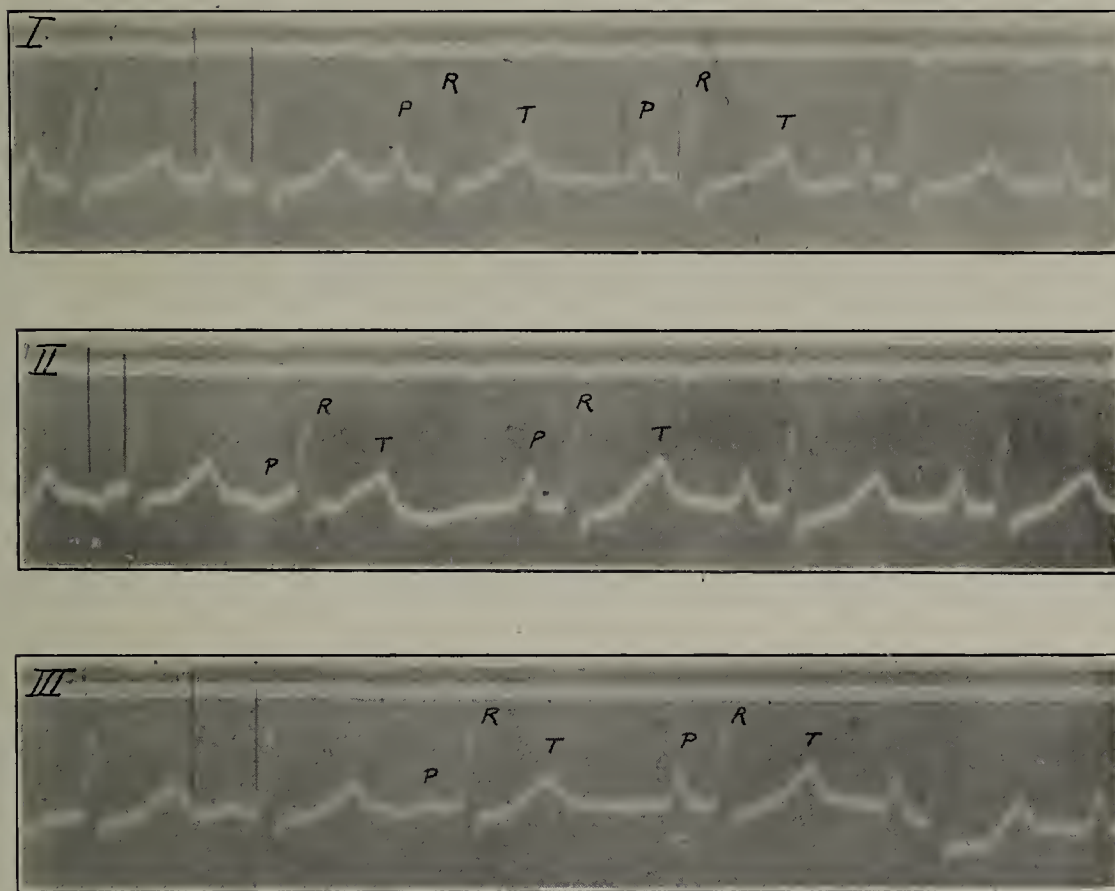


Fig. 15.

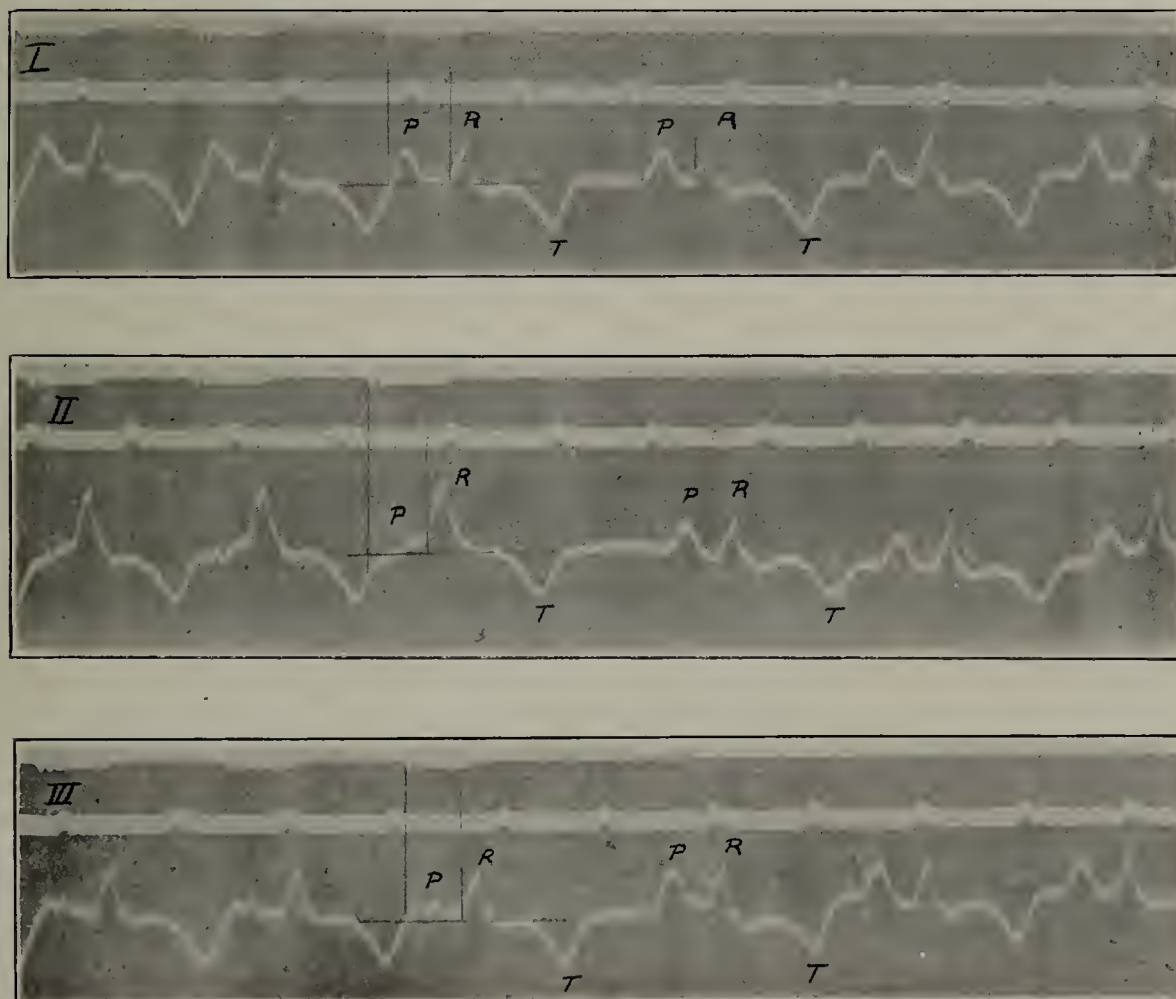


Fig. 16.

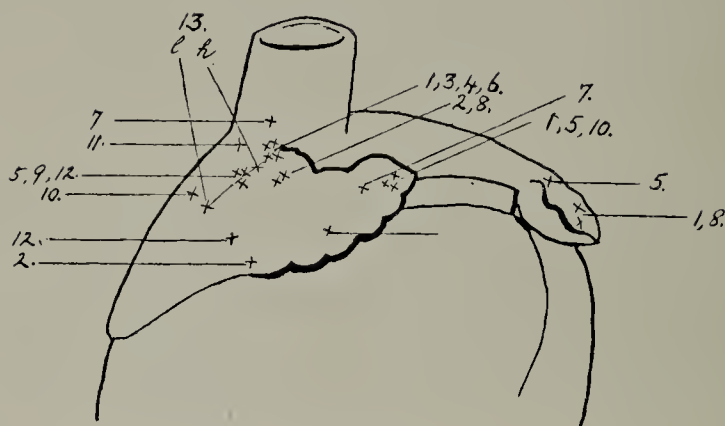


Fig. 17.

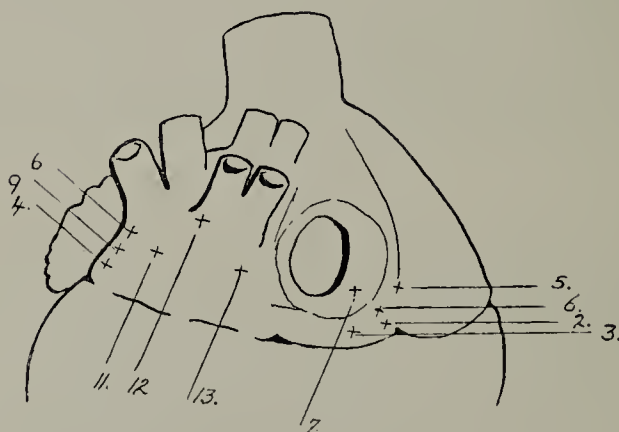


Fig. 18.

Fig. 17 and 18 (*Heart*, 1910, II, 29). Diagrams recording the points of stimulation adopted in a series of thirteen experiments upon dogs.

Fig. 19 (*Heart*, 1910, II, 27). A diagram showing the electric complexes of the auricular systoles in a series of thirteen experiments (natural size). The number of the experiments is indicated to the left. The point from which the auricular systole was propagated is indicated above. Each curve has been traced from the original photographs, examples of which are shown in Fig. 15 and 16. *N*=normal complex of the animal investigated. *S. V. C.*=that obtained from the superior vena cava (*h*=the upper point, and *l*=the lower point of excitation). *I. V. C.*=from inferior vena caval area (an asterisk is placed against curves obtained by internal stimulation). *P. V.*=from area of pulmonary veins. *C. S.*=from coronary sinus (internal stimulation). *A. B.*=from base of auricular appendix. *R. A.*=from right and *L. A.* from left auricular appendix. The lead was from right fore-paw and left hind-paw.

A comparison of the first two vertical columns demonstrates the constant resemblance of the normal auricular complex with that of beats excited in the superior vena caval area. A comparison of the first two with the third and succeeding vertical columns demonstrates the dissimilarity of the curves yielded by stimulation of all other parts of the auricular musculature.²¹

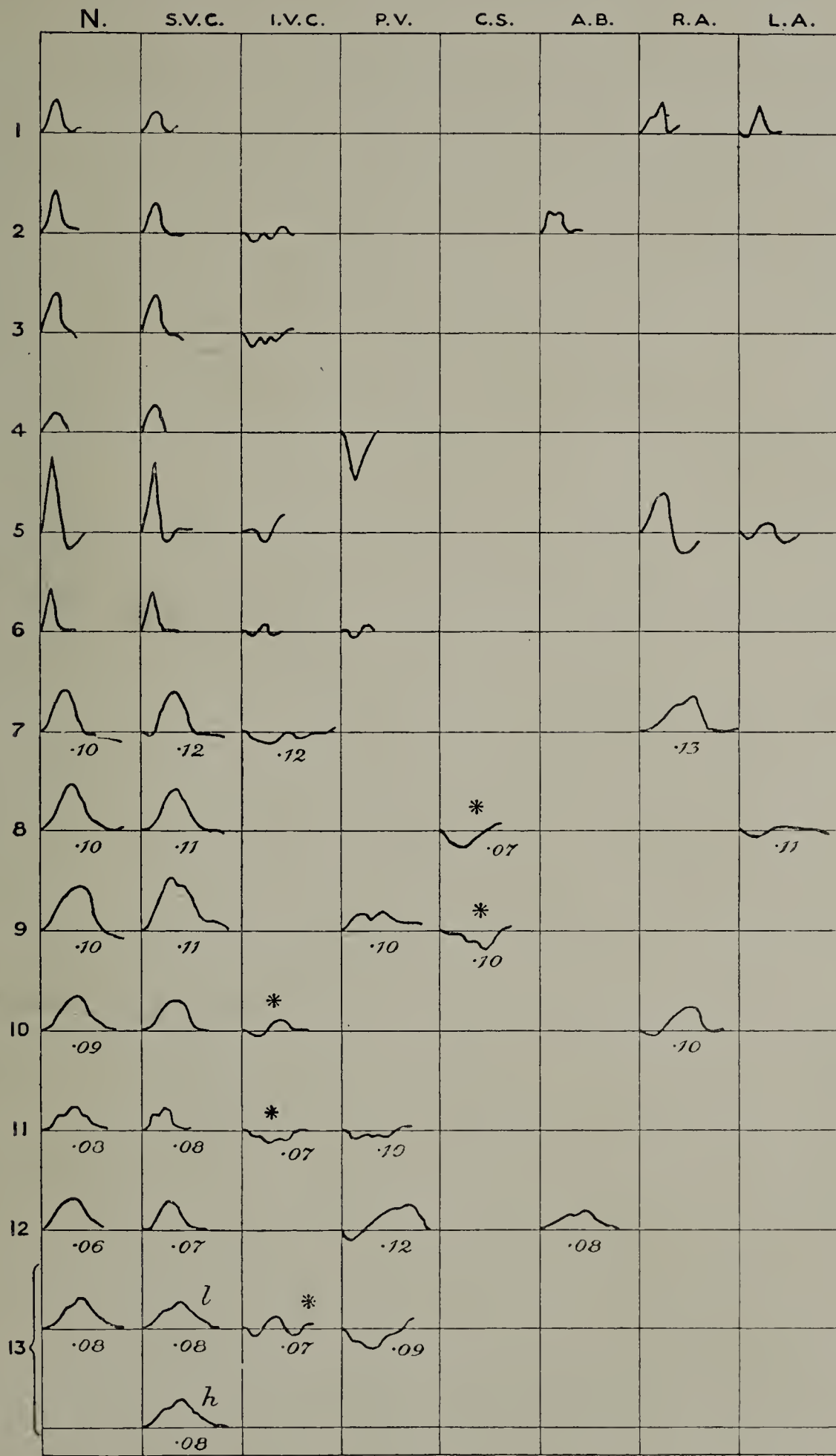


Fig. 19.

that the form of the electric curve is an index of the direction of contraction. The path taken by the normal contraction wave in the auricle is similar to that taken by a contraction wave, caused by artificial stimulation of the area surrounding the mouth of the superior vena cava ; which is tantamount to a statement that the points of origin of the normal and the excited contraction are practically coincident. It is also found that auricular curves of a similar nature are yielded by excitation of no other areas, but that in any given animal the type of curve obtained from inferior vena cava, coronary sinus, pulmonary veins, and other area, is perfectly distinctive, and bears no resemblance to the normal auricular curve. These facts are illustrated in Fig. 15-19. The observations are also of importance from a different standpoint. It is impossible to imitate the ventricular complex by stimulation of a single point or a small area of the ventricular musculature, and this is due to the fact that the impulse to the ventricle spreads to it through a system of special branching conductors. The observations upon the auricle strongly support the view that a similar system is not present in the auricle, but that the contraction spreads in all directions into the tissues which surround the pace-maker, and thence through the auricular walls to the auriculo-ventricular node.

The point of primary negativity.

When a strip of somatic muscle enters upon contraction, and the contraction starts at a given point, this point becomes negative relative to all other points. This is a law which has been well known to physiology since the time of its statement by Hermann. Now negativity is readily shown by a galvanometer, for, if the recording instrument is connected by means of two non-polarisable electrodes to two points upon the surface of a muscle, primary negativity of one contact is shown by a deviation of the shadow in a particular and known direction. When the contact connected with the lower end of the string of the galvanometer becomes relatively negative to that connected with the upper, the deviation of the string in the eventual photograph is in the upward direction. If the electrodes are applied direct to an auricle which is beating normally in an experimental animal, it is found that the sulcus terminalis of the right auricle becomes primarily negative to all points outside it, when this chamber enters upon its contraction ; and that, analysing the sulcus itself, negativity is first detected in a portion of it, which directly overlies the collection of nodal tissue described by Keith and Flack.^{17, 19 & 20} As a rule the primarily negative point lies in the immediate neighbourhood of the angle between the appendix and superior vena cava, and it is in this neighbourhood that the largest collection of peculiar tissue is usually found in the corresponding animals. The method adopted is illustrated by Fig. 20. The figure represents an outline of the base of the heart, viewed from the right side, and shows the superior vena cava above and the inferior vena cava below. The circles mark the point of contact of the electrodes. In the

particular experiment, which may be taken as a typical illustration of a number of experiments, leads were taken from the following pairs of points :— T^2 - T^7 , T^2 - T^5 , T^2 - T^3 . In each instance T^2 was connected to the lower end of the string, and the first movement of the string shadow was always in the upward direction. In each instance, therefore, T^2 was found to be primarily negative, that is to say primarily active as against the companion point. A further series of points were then tested as against T^2 , for instance, S^1 and S^3 , A^2 , P^1 and P^2 . T^2 was seen to be negative to each in turn. The general direction of propagation of the contraction wave in respect of a lead is indicated by a line and arrow joining two circles. In this manner it is possible to show in a given case that of all points on the sulcus the appendicular end of it first shows signs of activity, and that this point is also active at an earlier period than any points arranged in an approximate circle around it. Subsequent microscopic examination of the tissues actually investigated in the experiment demonstrated the presence of nodal tissue along the line indicated by the heavy dots, and the main collection lay in the immediate neighbourhood of T^2 . The order in which the leads were adopted is given in the table accompanying the figure. Of the points standing under the heading "lead" that which stands to the left was connected with the lower end of the string.

In addition to the leads already described, leads were also adopted from the upper and lower ends of the wound required to expose the auricle. And they were taken with very definite objects. The contacts included the greater part of the heart between them, and as the upper contact was joined to the lower end of the string, an electric curve of the whole heart beat was obtained in which the direction of the summits served as a control (Fig. 21). A repetition of the lead at or near the end of the experiment allowed a comparison of the heart action at the commencement and the end of the observations. In this way it could be shown, by careful comparison of the auricular portions of the curves, that the site of origin of auricular contraction remained unchanged during the progress of the experiment, an important consideration. The P variations of the first and second control curves presented a constant form.

The histological examination was accomplished by means of serial sections, transverse to the sulcus, of a number of blocks of tissue, each of which included one of the points of lead-contact on the sulcus. The width, depth and square area of the node in the sections, a selection of which is given in the accompanying table, were readily calculated.

The electric evidence as a whole appears to be quite conclusive that the heart beat originates in the immediate neighbourhood of the specialised and richly innervated tissue, lying in the upper stretches of the sulcus terminalis; the evidence in our possession in regard to the *primum movens* of the mammalian heart is consistent with the view that this point lies at the superior vena caval junction in the immediate neighbourhood of the sino-auricular node (described on page 2), and with this conclusion alone.

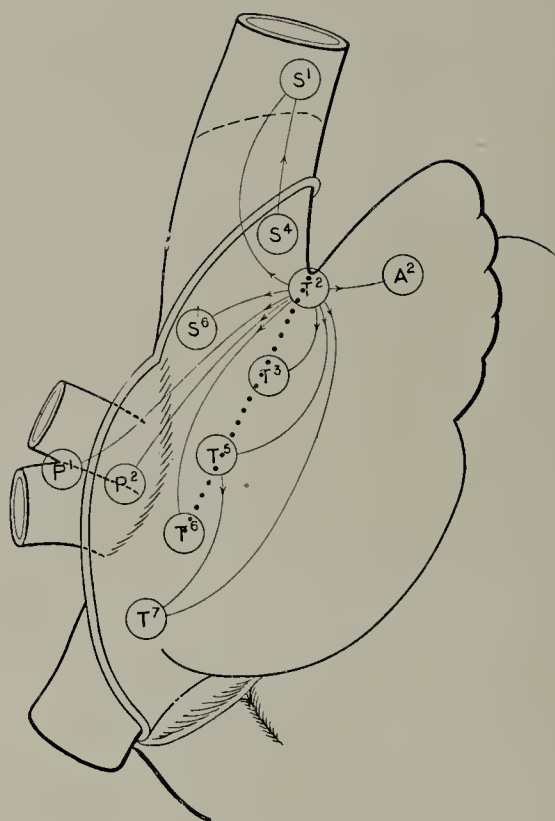


Fig. 20 (*Heart*, 1910-11, II, 158). A diagram showing the general directions of propagation of the contraction wave in the right auricle, as indicated by direct galvanometric leads. The point of primary activity (negativity) lies at T^2 . The distribution of the sino-auricular node, as ascertained histologically, is indicated by the dotted line.

<i>Lead.</i>	<i>Curve.</i>	<i>Direction of variation.</i>
Upper edge to lower edge of the wound gave a curve presenting the three usual upright summits P , R and T .		
T^2-T^7	.. Diphasic	.. upward, downward.
T^2-T^5	.. diphasic	.. upward, then downward.
T^2-T^3	.. diphasic	.. upward, then downward.
T^2-S^6	.. diphasic	.. upward, then downward.
T^2-A^2	.. diphasic	.. upward, then downward.
T^2-S^1	.. diphasic	.. upward, downward.
T^2-S^1	.. diphasic	.. upward, downward.
T^2-S^1	.. diphasic	.. upward, downward.
S^4-S^1	.. polyphasic	.. upward slight, downward slight, then markedly upward.
T^5-T^7	.. diphasic	.. upward, then downward.
T^2-P^2	.. not strictly diphasic	upward, then downward.
T^2-T^6	.. diphasic	.. upward, then downward.
Upper edge to lower edge of wound showed curves identical with those previously obtained.		
T^2-P^1	.. diphasic	.. upward, then downward.

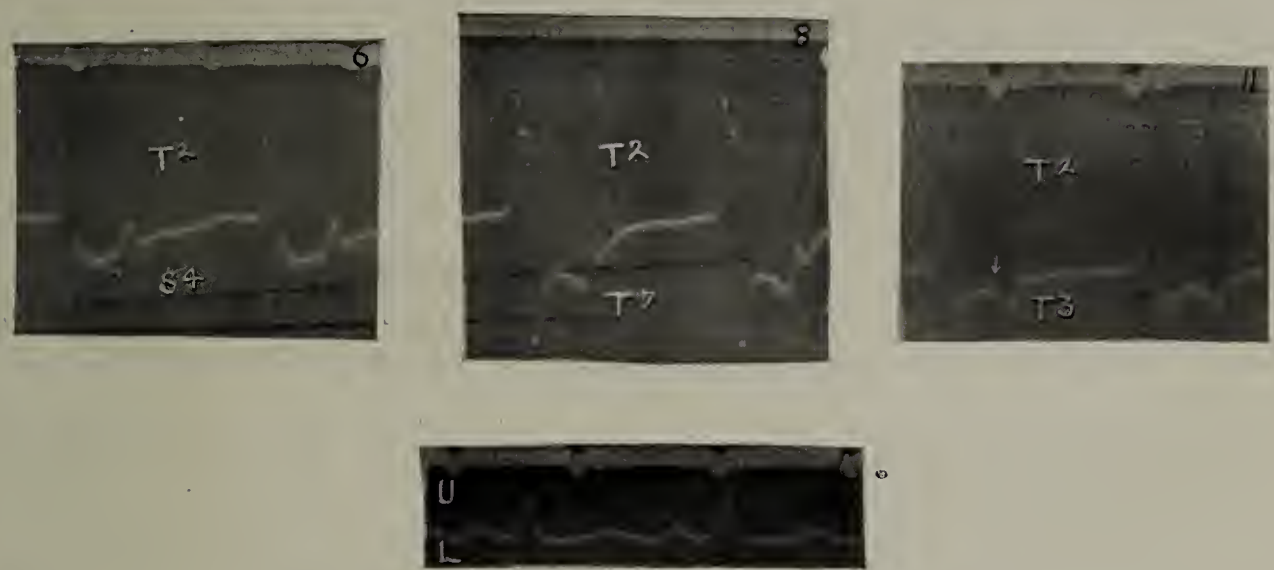


Fig. 21 ($\times \frac{2}{3}$) (*Heart*, 1910-11, II, 147, Fig. 8). Examples of curves obtained from another experiment of the sort described and obtained by means of direct leads. The corresponding contacts are marked in the four curves. The contact connected with the lower end of the string is placed above the curve in each instance. The fourth strip is a control from the upper and lower edges of the wound; it shows the three summits, *P*, *R* and *T*, directed upwards.

Table giving measurements of *S-A* node (from the heart outlined in Fig. 20).

Block, including area.	Distance in mm. from beginning of node.	Cross-section of node at selected intervals.			REMARKS.
		Width in mm.	Depth in mm.	Area in square mm.	
T ²	0.0	—	—	0.01 —	
	4.4	1.1 —	0.2	0.07 —	
	4.8	1.6	0.7 —	0.49 —	
	5.2	1.9 —	0.6 —	0.57 +	
T ³	6.0	1.4 —	0.6	0.40 —	
	8.8	0.8 +	0.8 .	0.35 —	
T ⁵	11.0	—	—	0.17 —	Node was dipping at this point and broke into two branches.
	14.6	0.8 —	0.4 —	0.13 —	
	16.8	0.9 +	0.2	0.09 —	
T ⁶	18.4	1.0 +	0.2 —	0.08 +	
	19.8	0.4 —	0.1 —	0.01 +	

20.4 mm. = total length of node.

The shape of the node resembled a *club*, of which the head was directed towards the superior cava.

Other Observations.

Observations upon the dying heart.—a) The *ultimum moriens*. In the moribund amphibian heart the last portion of the musculature in which contractions are visible is the pace-making sinus. A number of parallel observations have been undertaken upon the mammalian heart and they date back to the times of Harvey and Albrecht von Haller. It has been stated that the contractions are last observed in the terminations of the great veins,¹⁸ for example in the superior vena cava.⁹ On the other hand, they have been observed in the auricular appendix¹⁴ and in the pulmonary veins. The observations are coarse and probably largely unreliable. b) The beats of the dying heart are slowly conducted and the contraction waves may be seen to originate near the mouths of the great veins, and to be conducted from them.^{11, 14 & 18} The observations upon the dying heart cannot be held to have any great weight in deciding the site of the pace-maker. The heart is in a pronouncedly abnormal state, and it is well known that smaller nutritional disturbances may readily lead to a dislocation of the seat of impulse formation.

Warming and cooling the sinus or the tissues in its neighbourhood was employed by Gaskell and Engelmann, who found an increase or decrease of rate in the frog's heart according to the temperature to which the chamber was submitted. In the mammalian heart a similar method has been adopted by McWilliam,¹⁸ who states that the heart rate is accelerated when the tissues in the neighbourhood of the superior vein are heated. Adam¹ made further and more extended observations, stating that the point most sensitive to temperature change lies between the superior and inferior venous inlet, and rather nearer to the latter than to the former. He also reported the extension of the sensitive area to the base of the auricular appendix.

Now, in experiments in which heat is applied to the heart wall, it must be clearly understood that two entirely separate phenomena may be manifested. If the heat is applied over the site of the rhythm producer, a gradual quickening of the original rhythm will follow. If, on the other hand, it is applied at a distant point, and given that it is sufficiently intense, a new and rapid rhythm may be generated from this point. Adam stated that the most sensitive area lies nearer the inferior than the superior vena cava; he illustrated his contention by a curve which manifestly demonstrates the last phenomenon. It shows the origin of a new rhythm, for the acceleration is abrupt and not gradual, both in its onset and offset. The experiments in which cold is applied are more convincing, and some recently conducted observations⁵ seem to show that cooling in the neighbourhood of the sino-auricular node alone retards the rhythm.

Another method which has been adopted is that of weak electric stimulation of the areas of the auricle. Originally devised by McWilliam, who found the most sensitive point to lie at the mouth of the superior vena cava, it has recently received further attention from Flack,⁵ who comes to similar conclusions.

Excision or section.—In the cold-blooded vertebrate any injury which disunites the muscular continuity of sinus and auricle dissociates the rhythms of the two chambers, while that of the upper chamber is alone preserved.

Several attempts to obtain a corresponding result have been prosecuted upon the mammalian heart. Langendorf and Lehmann,¹⁵ using the perfused heart, found that amputation along the line of termination of the great veins leads to standstill of the amputated portions, while the stump continues its movements. The effect is not a result of interference with the vagus, for it takes place according to these writers in the atropinised heart. Erlanger and Blackmann,⁴ by using an ingenious clamp, have succeeded in damaging or separating certain areas of the auricular tissue. While they conclude that certain portions of the right auricle and the septum have a high grade of rhythmicity, they are unable to differentiate the normal point of impulse generation further than to state that in the majority of instances it lies at the mouths of the great veins. The experiments were carried out for the most part upon the perfused heart. So were also those of Hering,⁹ which though of a somewhat different nature may be noticed at the present time. Hering observed standstill of the supraventricular parts of the heart upon making cuts in the neighbourhood of the superior vena caval junction. In regard to the last experiment, it is by no means clear that this writer has excluded the possibility of a stimulation of the vagal nerve endings with which this area is richly supplied.

A serious criticism which applies to the majority of the experiments so far described under this heading is that they have been carried out upon the perfused heart, and it remains to be proved that the normal pace-maker is preserved under such circumstances.

Whether the validity of this criticism is admitted or not, the experiments as a whole show no more than that the pace-maker lies in the neighbourhood of the great veins. Hering has described a dislocation of the site of stimulus production as a result of the application of formaline to the inlet of the superior vena cava.¹⁰ Recently Jaeger,¹³ using the intact heart beating *in situ*, has cauterised and completely destroyed the sino-auricular node. He finds that the original and subsequent auricular rhythms are of the same rate. Flack observed no material alterations of rhythm after freezing or clamping the region of the node. Both in regard to these and previous observations, we may conclude that rhythmicity is considerable in several areas of the auricle, and that destruction of one area leads to the rapid assumption of the rhythm by an originally subservient centre.²²

The length of the pause succeeding artificially, excited beats.—Engelmann,³ in his researches upon the amphibian heart, found that a premature beat, resulting from excitation of the sinus, is followed by a disturbance of the fundamental or sinus rhythm, and that the pause following such a disturbance is equivalent to the interval between two beats of the fundamental rhythm. On the other hand, it was found that a contraction propagated by exciting the auricle itself is succeeded by a full "compensatory" pause. That is to say, the sinus rhythm is undisturbed; the pause following the interruption is of sufficient length to compensate exactly for the prematurity of the excited beat.

A similar method has been adopted in the study of the mammalian heart, and it has been found by several observers that the pause is of the shortest duration when the mouths of the great veins* are stimulated.^{2 & 8} But the records have not been convincing.† In the mammalian heart the length of the pause is very variable, being sometimes the equivalent of the interval between two natural beats, and having sometimes the full compensatory pause, but presenting much more frequently *an intermediate length*. As evidence of the seat of the pace-maker the method has proved inconclusive, and it must remain in this state until the intermediate length of pause is more thoroughly understood. (For further remarks, see page 130.)

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* Whether superior or inferior vena cava is not specified.

† Hirschfelder and Eyster¹² state that they have obtained full pauses from the mouths of the veins and shortened pauses from the auricular substance, and consider that the length of the pause is dependent upon the instant rather than the site of excitation. The curves which they give are imperfect.

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- ²⁰ WYBAUW (M. R.). "Sur le point d'origine de la systole cardiaque dans l'oreillette droite." Archiv internat. d. Physiol., 1910, X, 78-89.
- ²¹ These observations have been criticised by ROTHBERGER and WINTERBERG (Archiv f. d. ges. Physiol, 1910, CXXXV, 601). These writers believe that the curves may have been complicated by the break-shock of stimulation. This was emphatically not the case. Such deformity of the curves is usually avoided if threshold stimuli are employed, and if the electrodes are closely approximated. Any deformity which occurs, and it is but rarely present, is easily identified, and has been excluded in the construction of the original of Fig. 19.
- ²² More recently COHN & KESSEL (Archiv of internal Med., 1911, VII, 226-229) have published the results of excision of the node. They found, in the profused heart, that this procedure causes cessation of the heart beat in the majority of instances. These positive results are probably of greater value than the negative ones already quoted.

CHAPTER V.

THE ANALYSIS OF THE CARDIAC MECHANISM.

ARTERIAL PULSE CURVES.

STRICTLY speaking, arterial pulse curves are indices of the contraction of the left ventricle, but they may be utilised in the study of the disorders of the cardiac mechanism upon a broader basis ; for as we have no evidence of isolated contraction of right and left ventricles, they may be taken as indices of the contraction of the ventricular musculature as a whole.

Arterial curves in which there is variation from beat to beat, fall into two broad classes : (1) Those in which the pulse rhythm is regular, but in which the individual beats vary in amplitude (for example, respiratory variations of excursion, and the *pulsus alternans*, Fig. 22) ; (2) those in which the pulse rhythm is irregular.

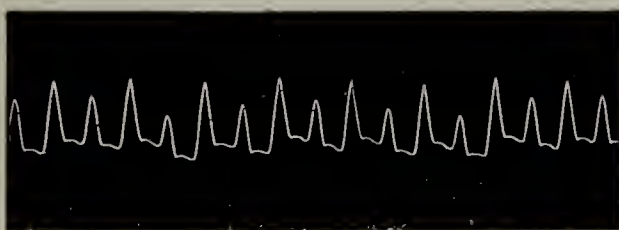


Fig. 22. An example of irregularity in the excursion of radial pulse beats. The sequence is regular, but alternate beats are of large and small excursion. A condition known as "*Pulsus alternans*."

To the study of the irregular arteriogram special attention must be devoted, for a great deal of information may be obtained from it ; in fact, it may be said that the mechanism of the heart may be identified in the majority of cases in which it is irregular by a careful examination of the radial pulse tracing alone ; in polygraph records it should always receive primary attention. But it must be understood that though such identification is possible, it has been rendered possible only by extensive observation. Thus, while the arteriogram often suffices as a clinical aid to diagnosis, *proof* of the presence of a particular heart mechanism ultimately depends upon the polygraphic and electrocardiographic examination. Of the facts which the arteriogram conveys, the most important are considered in the ensuing paragraphs. The propositions are stated categorically ; the evidence for them will be found in subsequent chapters.

The presence or absence of a rhythm dominating the movements of the ventricle.

In the analysis of irregularities of the ventricle, the first and most essential step consists in ascertaining the presence or absence of a *dominant rhythm*. By the term dominant rhythm the writer wishes to convey the meaning of a basal or fundamental rhythm which governs, more or less, the disordered ventricular movements. It is a rhythm which, in a case of irregularity, is disturbed either at its source or during the passage of its impulses from their site of origin to the ventricle; in the majority of instances the dominant rhythm arises in the pace-maker, and a large group of cardiac irregularities results from interruptions of the natural and regular flow of impulses from this point to the several cardiac chambers. So far as the study of cardiac irregularities has proceeded, the dominant rhythm of the ventricle, in any given case, may be asserted to be the one which is generated at the highest level of that portion of the cardiac tube from which impulses pass to the ventricle. This is usually the pace-maker, but occasionally the ventricle itself, or other points.

Where a dominant rhythm is present, those pulse beats which are preceded by the longest pauses are usually initiated by it; and in long stretches of curve such beats are most conspicuous in the tracing by reason of the amplitude of the lever excursion to which they give rise (Fig. 24 and 25).

If in an arterial curve, otherwise irregular, runs of four or more *prominent* beats occur from time to time, and the individual beats of the runs appear in perfectly regular succession, such beats are the outcome of impulses belonging to a dominant rhythm (Fig. 23).

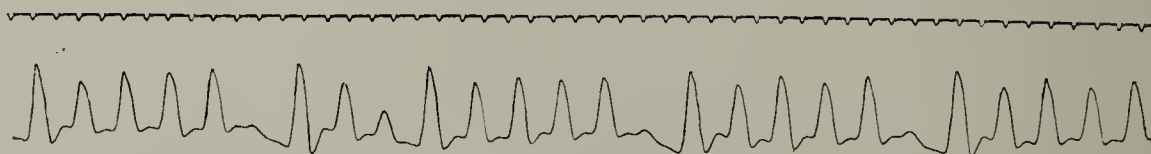


Fig. 23 ($\times \frac{2}{3}$). An irregularity in which groups of five regularly placed pulse beats occur from time to time. The beats belonging to the groups are of sinus origin and belong to the dominant or controlling rhythm.

A curve taken from a case during an attack of pneumonia. Alternation of the pulse, following premature contractions, is present. Recovery was nevertheless complete.

Further, if the curve consists of prominent beats separated by intervals which are frequently repeated, so that all such intervals fall sharply into a few groups (Fig. 27), a dominant rhythm is certainly present. It is also a rule that, if, from time to time, a period of irregularity of moderate length is duplicated (Fig. 24 and 25), the presence of a dominant rhythm

may be assumed, whatever be the nature of the irregularity. It is frequently found that in these instances all the intervals separating prominent beats are simple multiples of the shortest interval (Fig. 24 & 27), or that all are multiples of a common and large factor. Such a relationship is always conclusive evidence of the presence of a controlling rhythm, and also indicates the absence of disturbance of this rhythm at its source.

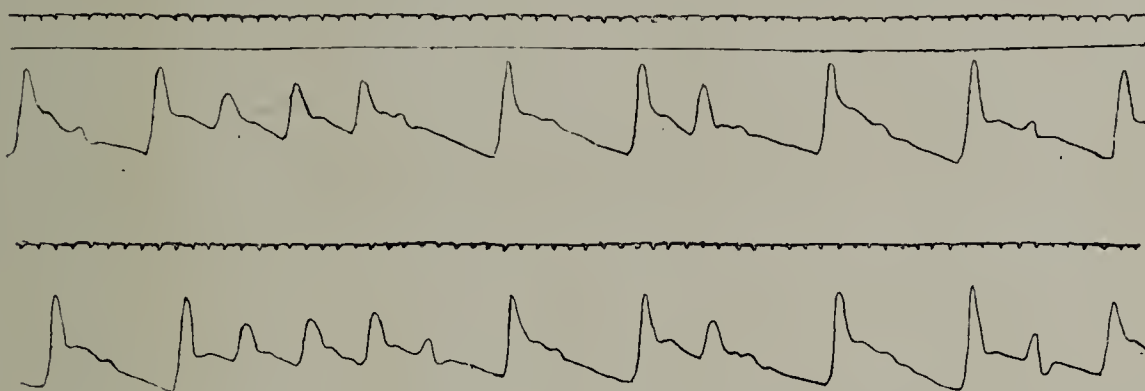


Fig. 24 ($\times \frac{5}{6}$). Two radial curves taken from a single patient. Illustrating the presence of a dominant rhythm. The lengths of the beats fall into a few categories; a period of irregularity of moderate length is duplicated. An irregularity due to *premature contractions** arising in the ventricle (the actual events were determined polygraphically). All prominent beats belong to the dominant rhythm (the third, fourth, fifth and sixth beats of the figure are representatives of the dominant rhythm, and they form together the only *run* of dominant beats in the figure).

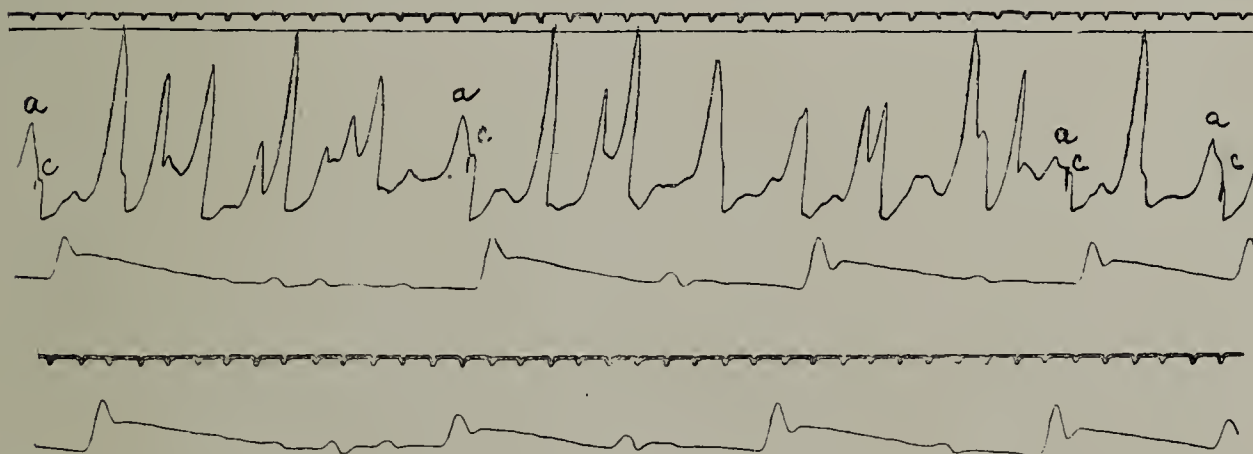


Fig. 25 ($\times \frac{5}{6}$). Two radial curves from a single patient. The upper one is accompanied by a venous curve (the two venous curves were identical in every respect, the lower one is therefore omitted). To illustrate the presence of a dominant rhythm, most of the prominent radial beats belong to it. The lengths of the intervals between prominent beats fall into a few categories. A period of irregularity of moderate length is duplicated. An irregularity resulting from premature contractions arising in the ventricle (the actual events were determined electrocardiographically).

* A *premature contraction* is one which appears too early in an otherwise regular sequence of contractions. Such contractions are fully considered in subsequent chapters.



Fig. 26 ($\times \frac{1}{2}$). An example of phasic variation of a dominant rhythm, independent of respiration. All impulses arose at the pace-maker (as shown electrocardiographically). The curve shows the characteristic and *gradual* passage from slow to fast and fast to slow rates. Intervals of varying lengths are arranged in orderly fashion in relationship to each other.

These facts are of special value in isolating and excluding a group of cases in which the pulse is completely irregular, a condition (auricular fibrillation) which is more closely discussed in Chapters XVII and XVIII.

The presence of regular sequences of beats.

When a pulse is regular for long periods it may be taken that the rhythm is promoted by impulses arising in a small and limited area of the musculature, and probably from a single point. And this statement holds true whether the rhythm originates in the normal pace-maker or not.

Observation allows a further statement. Where a number of pulse beats are found, for example six or more, and they are equal in amplitude, and regular in their succession, the impulses from which they originate are generated at a single focus, whether the rhythm of which they are an expression is dominant or interrupting. An example of an interrupting rhythm of 10 beats (arising from a single point in the ventricle) is shown in Fig. 27 *d*.

Phasic variation of the dominant rhythm.

The reverse statement, that a rhythm arising from impulses generated at a single focus is regular, is not necessarily true. For if such a rhythm is built up at the pace-maker it frequently happens that it shows periodic variations of rate. It is often possible to identify these variations in the arteriogram, if simultaneous observations are undertaken of the respiratory events, as the variations are often determined by the breathing. They consist of a gradual waxing and waning of pulse rate, repeated from time to time, and at definite intervals which are related to the respiratory cycles. Phasic variations of a dominant rhythm which are independent of respiration also occur (Fig. 26), but cannot be identified with certainty without the aid of records from other heart chambers. (Variations of this character are more especially considered in Chapter XXI).

The rate of the dominant rhythm.

The rate of the dominant rhythm, usually ascertainable in the arteriogram, is of importance in studying disorder of mechanism in that it offers a clue to the source of such a rhythm, though it can rarely be held as decisive in this respect. Now the dominant rhythm may have its origin in the normal pace-maker, and under these circumstances the rate usually approaches 72 to the minute; but the limits of variation are great and pass from 30 to 240 to the minute. The dominant rhythm may have its origin in the ventricle, and under these circumstances its rate approaches 30 to the minute (the known limits in man are from 0 to 63 to the minute). Not infrequently a new rhythm of pathological type may prove dominant, and such rhythms may arise at many points in the musculature. The rate of these new rhythms is variable and the limits are at present ill-defined but probably approach 130 to 300 per minute.

Thus if the dominant rhythm has a rate approaching 70, there is presumptive evidence that it is generated in the pace-maker. If the rate is approximately 30, the rhythm is usually of intrinsic or idio-ventricular origin. Lastly, if the rate lies constantly and continuously between 130 and 300 it should awaken a suspicion of the presence of new or pathological impulse formation.

The presence or absence of disturbance of the dominant rhythm at its source.

This is of importance, as we shall subsequently find, in determining the proximity of the source of the dominant rhythm and the point of origin of events disturbing the sequence of the contractions to which such a rhythm gives rise. And it may be taken as an axiom that, *per se*, hindrances to the passage of an impulse from its source never materially influence the dominant rhythm at the source. A demonstrable interference with the rhythm at its source is the result either of intrinsic causes or is a consequence of a premature contraction started in some other portion of the heart musculature, the effects of which are reflected to the source of the dominant rhythm. And it is found experimentally that a disturbance of the dominant rhythm of the last form is the more liable to occur the more nearly the seat of extraneous impulse formation approaches the source of the rhythm which suffers.

The absence of interruption of a dominant rhythm at its origin is clearly demonstrated in an arteriogram if the rate of the dominant rhythm is known and if the beats resulting from such a rhythm fall without exception upon ordinates evenly "spaced" at such a rate (Fig. 27).

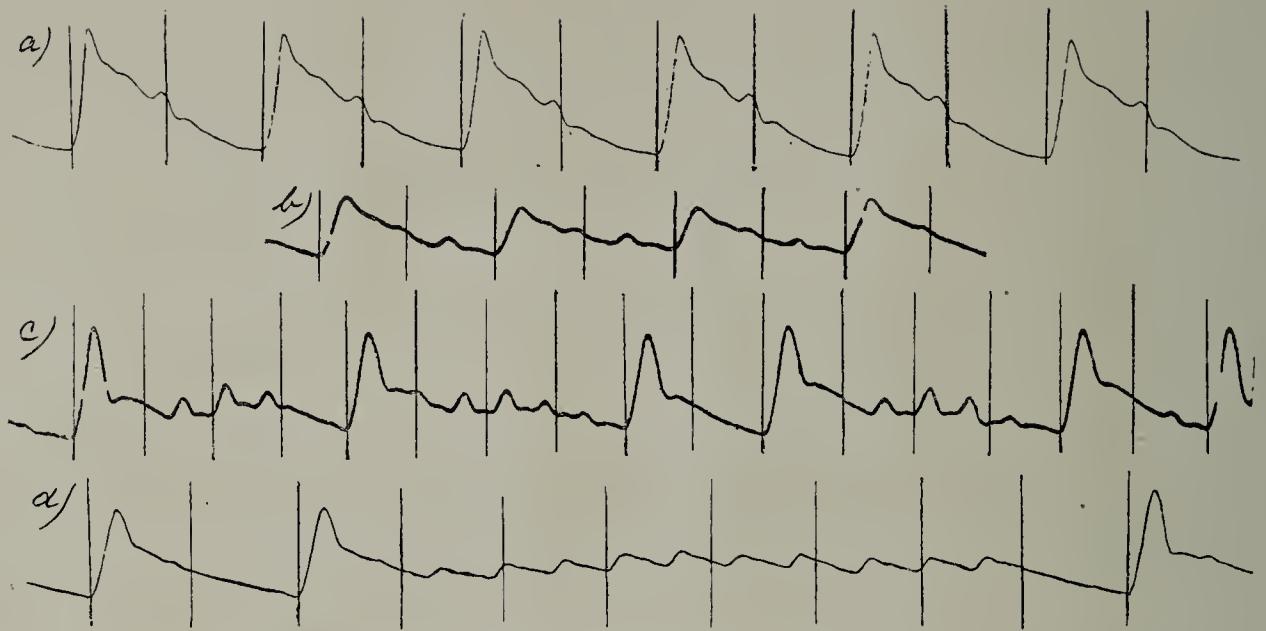


Fig. 27 ($\times \frac{5}{6}$). Four examples of irregularity taken from a single patient. The arrhythmia resulted from premature contractions arising in the ventricle. Ordinates are drawn at equal distances upon each separate curve. The curves illustrate a step in the analysis, which may be termed "spacing." The dominant impulses were shown to be arranged in the fashion depicted in the figures by the vertical lines, by means of both polygraphic and electrocardiographic curves. Curve *c* is an example of a grossly irregular pulse, but a pulse which is not absolutely irregular. All the intervals between prominent beats are simple multiples of the shortest of these intervals. There is no disturbance of the dominant rhythm. Curve *d* shows a similar phenomenon and proves the absence of disturbance of sinus rhythm by a succession of 10 or 11 premature contractions arising in the ventricle. The first dominant beat following the row of fast beats falls precisely at the expected point. (Fig. 115 is from this case.)

The presence or absence of interference with the dominant impulses during their passage to the ventricle.

Provided that all beats of the dominant rhythm, usually that of the pace-maker, affect the arterial curve, the presence or absence of such interruption is determined with facility in the instance of an irregularity, in which the rate of the dominant rhythm is known, and in which beats of the dominant rhythm are determinable from point to point in the curve. For if disturbance of the ventricular sequence can be shown, and the interferences with the dominant rhythm at its source can be excluded by spacing, such disturbances can only occur as the result of interruption to the passage of the impulses. And the inability of the impulses to reach the ventricle may result from one or two causes. It may be dependent upon an abnormality of the function of the tissues conducting such impulses, or it may result from the refractory condition of the ventricular musculature as a result of its premature contraction (Fig. 27 *a*); that is to say, it may result from the presence of premature beats. Premature beats which

interrupt an otherwise regular rhythm are readily recognised in an arteriogram when they are propagated to the ventricle and affect arterial pressure. They lie low in the curve and, commencing earlier than the anticipated beat, are usually followed by more or less prolonged pauses (Fig. 27*a*). They are usually unaccompanied by a predicrotic wave (Fig. 27*a*). Their presence is established, when they fail to effect the arterial curve, by means of auscultation or by apical or electrocardiographic tracings.

The determination of the lengths of the pauses following single or successive premature beats.

The measurement is of value in indicating the source of the premature contraction and will be referred to in more detail at a later stage. At present the following categoric statements may be made in regard to such pauses:—

1. If the pause is of a length equivalent to that between two beats of the dominant rhythm the source of the premature beat and the source of the dominant rhythm probably lie in the immediate neighbourhood of one another (Engelmann).²

2. Further, it may be said that, on the whole, the greater the distance between the points at which dominant rhythm and premature contraction arise, the longer, relatively, is the pause which follows the premature beat.

3. And finally it may be stated that if the pause is of full length, that is to say if it compensates for the curtailment of the previous pause, and re-establishes the spacing of the dominant rhythm, in all probability the premature contraction has arisen in the ventricle.*

Complete irregularity of the pulse.

When a pulse is completely irregular, that is to say when the intervals between beats are of very varying lengths, when there is no regular lengthening from beat to beat or subsequent regular shortening from beat to beat, when a phase of irregularity fails to repeat itself periodically, when the lengths of beats do not fall into a few simple categories, a dominant rhythm is absent, and the heart's mechanism is one known as fibrillation of the auricle (Fig. 28).

The value of the arteriogram to the student of the cardiac mechanism can hardly be exaggerated. It forms the foundation of the analysis of irregularities of the heart. And we owe to Wenckebach a great debt

* Premature contractions arising in the auricle occasionally give rise to this picture, but the instances are exceptional.

for the invaluable observations which he has made upon it.³ His critical analysis of measured arterial curves, published in his now classical treatise,⁴ laid the basis of the new studies. His insight led him to a differentiation of a large number of the more important cardiac disturbances from an examination of these curves alone.

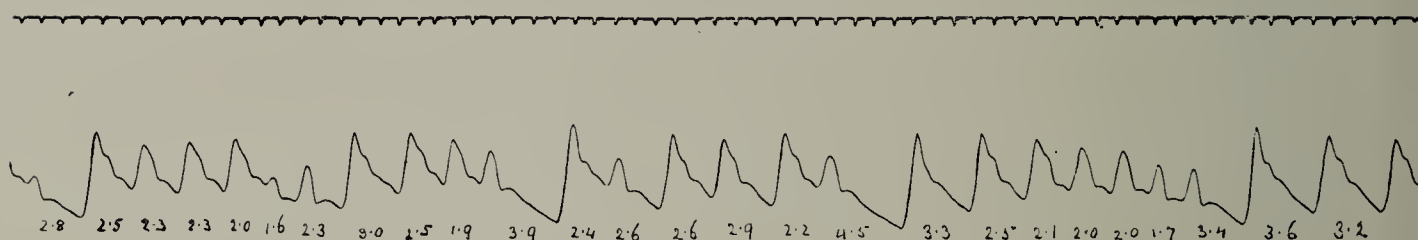


Fig. 28 ($\times \frac{2}{3}$). Complete irregularity of the pulse. From a patient with fibrillation of the auricle. The intervals from pulse beat to pulse beat show the extreme variation characteristic of the condition. The lengths of the beats are marked in fifths of a second below the curve.

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CHAPTER VI.

THE ANALYSIS OF THE CARDIAC MECHANISM *POLYGRAPHIC CURVES.*

IT has been stated that the venous pulse is an index of intra-auricular pressure, and we have seen that it shows the moment at which auricular systole commences; in this lies its chief significance. The exactitude of the indication is known by actual experience to be sufficient for the purposes for which it is employed. The arterial pulse, carotid or radial, acquaints us with the moment of onset of ventricular systole. It is by the utilisation of the simultaneous records that the chief and ultimate aim of polygraphic work is accomplished, and this aim has for its purpose the determination of the onsets of the auricular and ventricular systoles in a given case and their time relationships to one another. The analyses afforded by the polygraph are of the utmost value in promoting our knowledge of the clinical pathology of heart disease; and as students of this subject we are immeasurably indebted to Mackenzie who established this method.

The allowances for transmission intervals in arterial and venous curves have been described, and we may now examine the means employed in fixing the instants in question in routine practice. The *modus operandi* adopted is that which was originally devised for this purpose by Mackenzie.

The fixation of "c" in the jugular curve.

In most instances the radial curve is taken as the standard from which all measurements are made, for the artery at the wrist presents certain natural advantages, which render it the most convenient and serviceable point of arterial pulsation. The radial upstroke, consequently, is taken as the standard for the instant of onset of ventricular systole, and it is first corrected from a simultaneous carotid curve in order to allow for the difference in transmission time from heart to radial and heart to carotid respectively. In this way a point is obtained upon the radial curve a little (about .1 sec.) previous to the upstroke, which represents the time at which the upstroke of the carotid occurs in the neck. In simultaneous venous and radial curves this point is transferred to the venous curve, and in the latter it gives the instant representing the onset of ventricular systole. (These measurements may be made in Fig. 30.) In brief, the measurements fix the upstroke of the *c* wave and permit of its identification. The two

measurements which are involved are usually accomplished by a single transference. A short strip of venous and radial curves is taken; the clock is stopped, and index marks are written; short strips of carotid and radial curves are then obtained. The interval ($C D$) between two radial upstrokes, one lying to the left (Fig. 29 C) and one lying to the right (Fig. 29 D) of the index marks is measured. The carotid upstroke (B) corresponding to the radial upstroke to the right of the stops, is fixed and a point (A) is determined upon the venous curve which is separated from it by the distance ascertained in the previous measurement. The transference of the measurement is justified when the clock is running at the

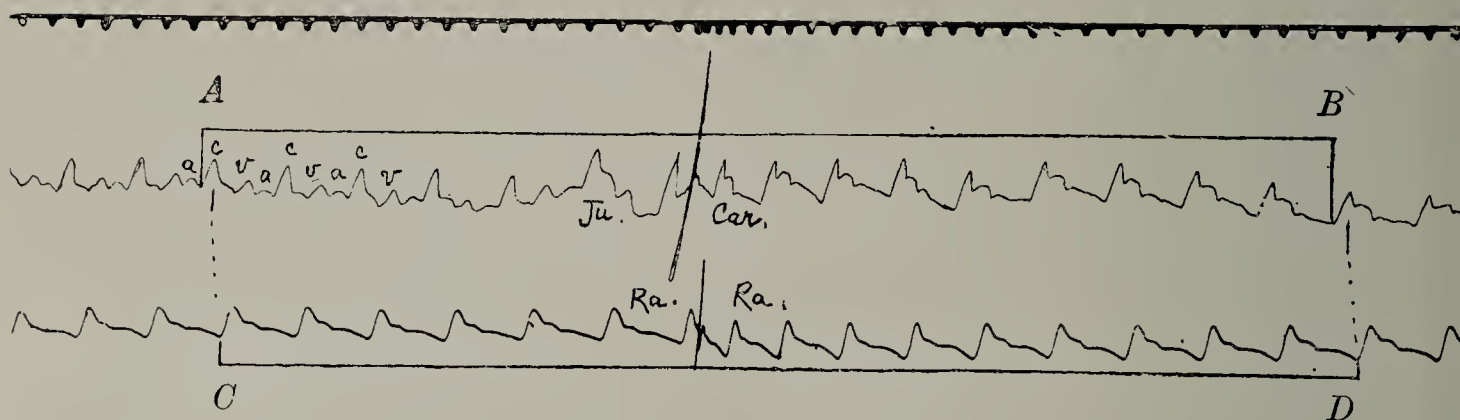


Fig. 29. Polygraphic curve, illustrating a method of identifying the c waves which is adopted in practice. Simultaneous radial and jugular curves are shown to the left, and simultaneous carotid and jugular curves to the right of the central index marks. The transferred measurement is indicated by means of the lines AB and CD .

same rate both to the right and left of the index marks, and when the curves to right and left lie at the same level, and under these conditions alone. The question of level has always to be considered in using levers which write through an arc of a circle. Where accurate measurements of intervals are required, correction from the actual index marks is absolutely essential, and the points obtained should be transferred to the time marks. Where the waves of the jugular curve are of comparatively simple form and are clearly inscribed, a single measurement from the index mark to the upstroke of a radial beat and a transference of this distance from the upper index mark to a point in the venous curve is usually sufficient to permit the recognition of c ; for the upstroke of this wave will lie at approximately .1 sec. to the left of the point ascertained. The wave c is used as an *index* of the onset of systole; the *actual* instant of onset may be obtained from a simultaneous cardiogram, mechanical or electric, and may be marked upon a venous curve by allowing for the transmission intervals from heart to aorta (presphygmie interval) and aorta to carotid. In practice this procedure is rarely required.

The fixation of "a" in the jugular curve.

In a venous curve, where a definite group of three clearly inscribed waves accompany each cardiac cycle and where one of these waves lies directly in front (to the left) of the point which marks the upstroke of *c*, this wave is known to be *a* and its upstroke represents the onset of auricular systole. The point of onset can be ascertained in most instances with certainty, for as a rule the wave is a prominent one and clearly inscribed from start to finish. In instances where the wave shows division, or where the upstroke lies at a distance of more than .2 sec. from the onset of *c* greater caution should be adopted in marking the curve. Too much stress cannot be laid upon the necessity of observing the constancy of the shape and position of such a wave from beat to beat or from one respiratory cycle to the next. In all cases it is essential that similar strips to those marked should be obtained, for without these duplicates the interpretations are as a rule valueless. Where there is any doubt as to the limits of *a* the observations should be checked from measurements of *a* waves as they occur at other points in the neck, from similar waves upon the apex curve, or lastly from the evidences of auricular contraction in electrocardiograms.

The "As-Vs" interval.

The interval *a-c*, measured from the commencements of the corresponding waves and taken as an *index* of the *As-Vs* interval, the true interval separating the commencements of auricular and ventricular systole is customarily .1 to .2 sec. It is usually slightly longer than the corresponding *As-Vs* interval as shown by electrocardiograms, and probably for this reason, that there is a greater delay in the appearance of *c* than of *a*. The increased length of the interval as compared with the more exact *P-R* interval of electric curves does not appreciably detract from its value as an index. The *a-c* and *P-R* intervals never differ from each other by more than .1 sec. (generally the difference is far less), consequently a prolongation of the *a-c* interval to .3 sec. or over is a definite indication (an indication which can be confirmed electrocardiographically) of a prolongation of the *As-Vs* interval. Even greater reliance may be placed upon a notable change in *a-c* interval from beat to beat in one and the same case.*

The value of the fixation of the summit of "v."

The upstroke of *c* is determined and checked from the arteriogram. The onset of *a* cannot be checked in a similar manner, and in the absence of electrocardiograms the measurement of *a-c* intervals should be circumspect. The accurate determination of the limits of ventricular systole

* Lewis. *Quart. Journ. Med.*, 1911 (April).

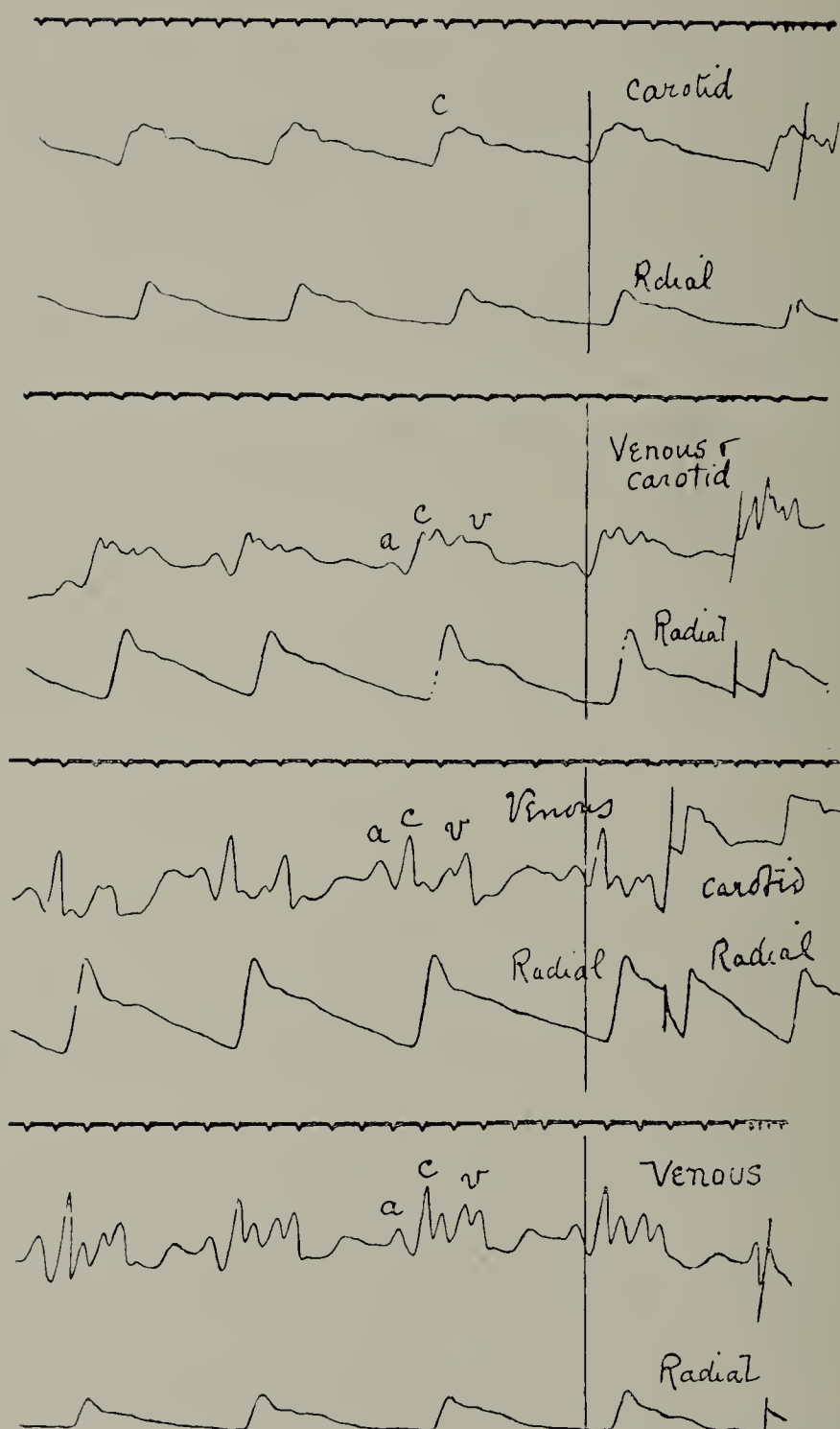


Fig. 30. This figure includes four polygraphic tracings taken from a single patient. The lowest curve in each strip is from the radial artery. The upper curve in all tracings is from a single and fixed point in the neck. The differences in the neck curves are dependent upon the pressure at which the receiver was applied. In the uppermost curve the pressure was heavy; in the lowermost curve light; in the middle curves moderate. The figure shows transitions from the arterial to the venous type of curve as pressure upon the neck is relaxed and the receiver recedes from the artery. The first tracing exhibits a curve of purely arterial character; the second tracing demonstrates a small *a* wave directly to the left of the carotid upstroke; the third tracing shows an almost perfect, the fourth a fully developed, phlebogram. Vertical lines have been drawn at approximately correspondent points in the four tracings.

The figure illustrates a simple method of proving the presystolic time relationships of the wave *a*.

is of importance in cases where there is a possibility of mistaking v for a or in instances in which a and v are suspected of coinciding (as in cases where the heart beat is rapid and v is insignificant, or as in cases where the a - c is prolonged).

The end of ventricular systole is represented in the carotid curve by the dicrotic dip, and in the jugular curve by the apex of v . The summit of the latter is synchronous with a point lying a little (usually $\cdot 1$ sec.) to the right of the bottom of the dicrotic depression in the carotid. In the radial curve the measurement may be made from the depression of the dicrotic itself. The summit of v may be also checked by measuring the length of the systole in a single carotid beat and transferring it to the jugular curve. The measurement is of great practical value and the position of the summit of v , and its relationship to the offset of systole, are of considerable constancy, within the practical error of measurement.*

The utilisation of measurements of the length of ventricular systole is illustrated by the accompanying curves (Fig. 31 and 32).

THE INTERPRETATION OF CURVES IN WHICH THERE ARE DISTURBANCES OF SEQUENCE.

The chief interest in venous curves centres around the identification of auricular systoles, and in the interpretation of curves, it is imperative that no wave be marked a until all other sources of wave production at the instant of its occurrence are excluded, or until the factors operating at such an instant are found to be insufficient to account for the full wave which appears.

The interpretation of abnormal curves depends to a great extent upon our knowledge of experimental irregularities. Where the usual form of v is modified from point to point in a curve, and where an unusual and synchronous event is observed in the arteriogram, the factors inducing such a variation of v should be diligently sought. If the alteration of v is in the direction of its exaggeration, it may be suspected that an auricular systole has fallen during the ventricular systole. And such a view is confirmed if the time relationships of the suspected v vary from the customary relationships (Fig. 31).

* The practical value of the measurement may be illustrated by reference to a recent paper by Fulton, Judson and Norris (*Amer. Journ. med. Sci.*, 1910, cxi, 339-348). These authors report what, if they establish their records, must be regarded as a case of hereditary heart block, in the broad sense. They give curves from three members of the same family. In one of the tracings the pulse is beating at 75 and the accompanying jugular curve shows a double wave following c . The authors conclude that the second peak of the double wave is auricular. But the fact that its summit falls at the opening of the tricuspid valves and that, regarded as an a wave, it is premature, precludes the acceptance of their interpretation, (cp. *Brit. med. Journ.*, 1911, i, 593).

Prominent peaks.

When, from time to time, extremely prominent peaks occur in the jugular curve, and when such peaks have an amplitude which is notably in excess of that of any of the peaks accompanying cycles of the normal and regular rhythm of the same case, it is probable, in the absence of marked variation in the amplitude of venous waves as an accompaniment of respiration, that at such points auricle and ventricle have contracted together, and that the auricle, *in lieu* of emptying itself into the ventricle, has created a sudden rise of venous volume.^{5 & 6}

Frequently, when these exaggerated peaks occur outside the limits of the systoles of the *rhythmic* ventricular beats, it will be found that an auricular systole is anticipated at this point, and usually evidences of an abortive ventricular contraction will be discovered in the radial, apex or electrocardiographic curves (Fig. 35).

And again, when such an exaggerated wave occurs and evidences of the presence of a relatively abortive ventricular systole are found, *even* though an auricular systole is not anticipated to complete a regular auricular rhythm, it is probable that it has occurred. For a relatively weak ventricular contraction fails to give rise to an exaggeration of its accompanying *c* wave, but usually yields a *c* wave which is diminished in extent, and some other factor must be found to account for its exaggeration. And

Fig. 31. A jugular and a radial curve from a patient, the subject of early or premature contractions arising in the auricle. The figure is given for the sake of the comparison of the third venous wave in the first and second cycles. It is to be noticed that in the first cycle it is less prominent than in the second and third cycles. In the first cycle it constitutes a true *v* wave, in the remaining cycles it is a complex of *v* and *a* waves. The difference in mechanism of the separate cycles, suspected from the appearance of the waves, is strengthened by the notable variation in the lengths of the radial beats. The respective mechanisms are identified by careful fixation of the summits of the waves in relationship to the limits of ventricular systole (*E, E*). In the first cycle the summit of the third wave falls precisely at the end, in the second cycle a little to the left of the point representing the termination of ventricular contraction. The measurement shows the first to be a normal cycle, and the second to be a cycle complicated by a premature auricular wave; an interpretation which received complete confirmation in this instance from the corresponding electrocardiographic records.

Fig. 32, 33 and 34. From a patient, suffering from mitral stenosis, in whom a slight grade of heart-block was demonstrated (electrocardiographically). The waves *c* were readily determinable, and it remained to ascertain the nature of the waves marked with an asterisk. The fixation of the limits of ventricular systole (*E*) clearly showed their independence of this systole, and as the waves in question were prominent it could only be concluded that they resulted from auricular contractions. That this was actually the case could be clearly shown from companion curves of transitional types (Fig. 33 and 34). The wave could be traced from its normal position (to the immediate left of *c*) to a point more removed from *c* (compare the cycles in Fig. 34). As *a* fell further and further back upon the *preceding* ventricular systole it came to occupy a position in which its differentiation from *v* would have been difficult or impossible (Fig. 33) in the absence of other evidence.

Note the progressive increase in the height of *a* as it falls further back upon the preceding ventricular systole in these figures.

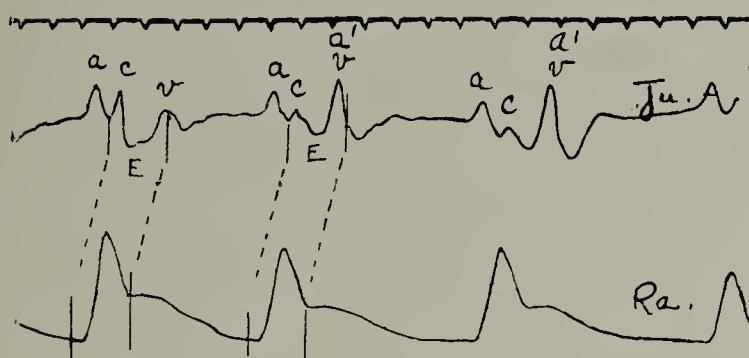


Fig. 31.

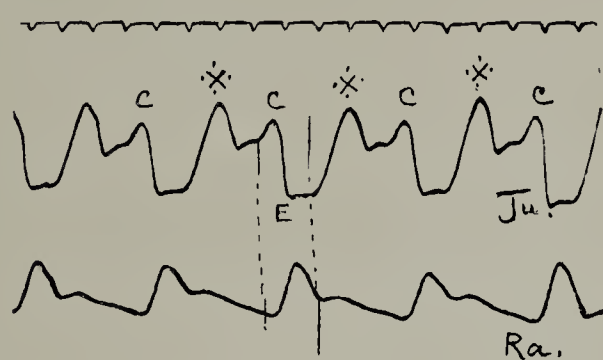


Fig. 32.

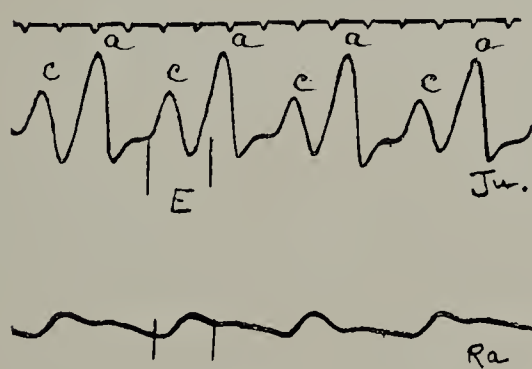


Fig. 33.

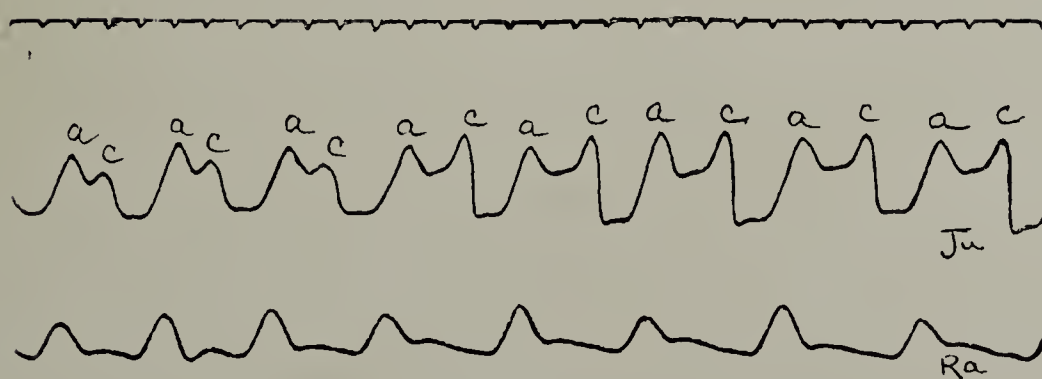


Fig. 34.

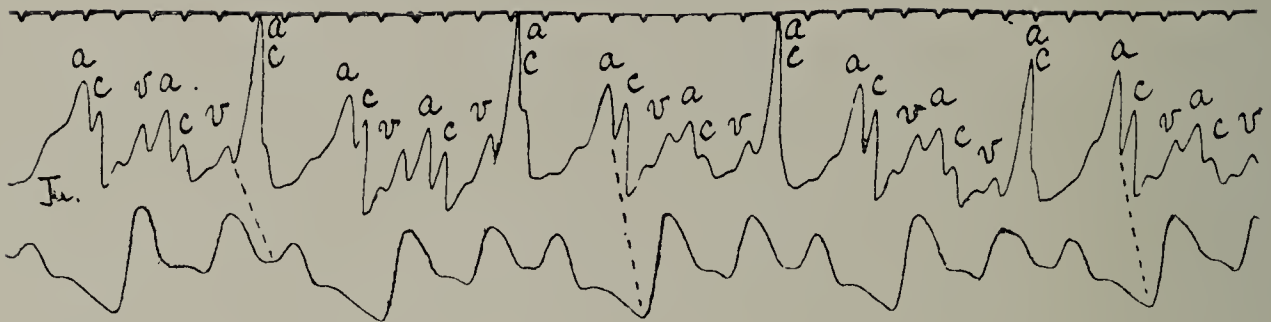


Fig. 35. Polygraphic curve from a patient exhibiting premature beats arising in the ventricle. The venous curve shows four exaggerated waves (a_c) each of which occurs at a point at which an auricular wave is anticipated. An abortive radial beat appears at a precisely corresponding instant in each instance. The simultaneous contraction of auricle and ventricle results in the prominent wave a_c .

the view that simultaneous contraction of auricle and ventricle has occurred at such a point will be confirmed if a disturbance of the dominant rhythm is found accompanying it (Fig. 36); but it may be strongly suspected even though no such disturbance occurs.

Thus exaggerated waves of the kind discussed are encountered outside the limits of regular ventricular systoles when a premature ventricular contraction coincides with an expected auricular systole (Fig. 35); and also when a premature ventricular systole coincides with a premature auricular systole (Fig. 36) (a form of premature heart contraction which will receive subsequent attention). Similar exaggerations occur *during* the limits of regular ventricular systoles, and under several circumstances. For a premature auricular contraction may be so early that it coincides with the preceding ventricular systole, or the *As-Vs* interval of a normal and regular rhythm may be so far increased as to lead to a similar result (Fig. 33 and 37); and the latter is especially the case when the heart rate is increased. It is questionable whether simultaneous auricular and ventricular systoles occur as a result of increased rate in the absence of damage to the conductive functions of the junctional tissue, for with increased heart rate there is usually a proportionate increase in the rate of conductivity and a consequent decrease in the *As-Vs* interval. But when slight conduction changes are present, the auricular contractions frequently fall back upon the preceding ventricular systoles, and in the absence of actual prolongation of the *a-c* interval. It is a frequent phenomenon in cases of pathological impulse formation in the auricle, where successive and premature contractions give rise to paroxysms of tachycardia (Fig. 38). The auricular systole and ventricular systole often fall together in cases of dissociated auricular and ventricular rhythm and a similar exaggeration of the accompanying venous curves results (Fig. 40).

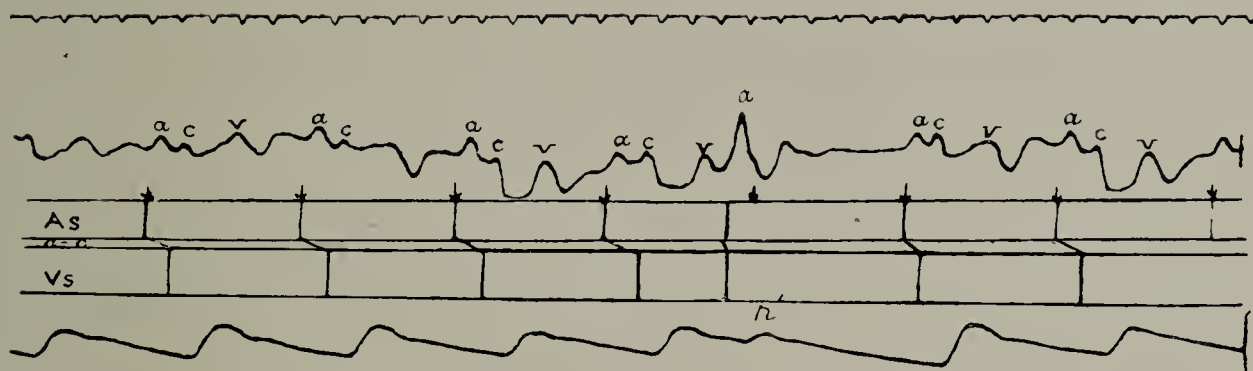


Fig. 36 (*Mackenzie, Quart. Journ. Med.*, 1907-8, 1, 142, *Fig. 16*). A polygraphic curve showing a single exaggerated wave c'_1 as a result of simultaneous and *premature* contraction of both auricle and ventricle.

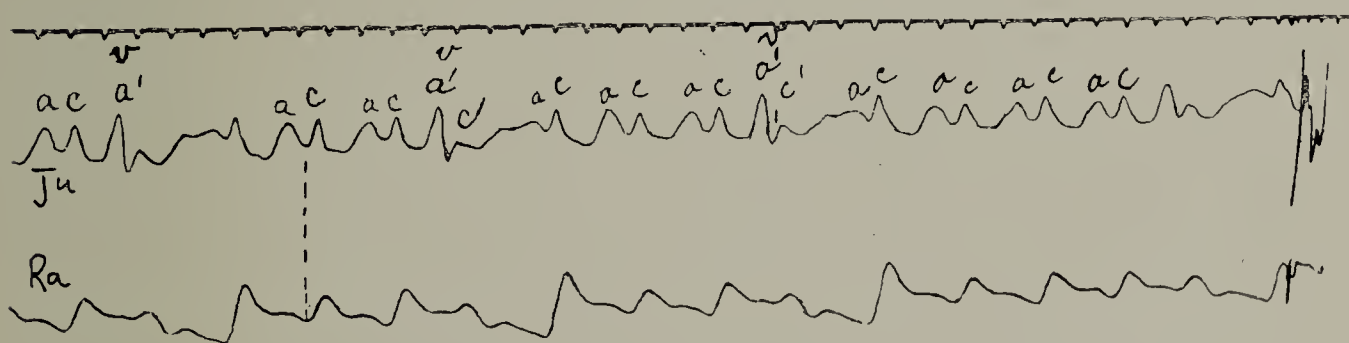


Fig. 37. A polygraphic curve showing three prominent waves $\frac{a}{v}$ resulting from premature contractions of the auricle; the exaggeration is rarely so great, in the event of a and v coinciding, as in the event of a and c being synchronous.

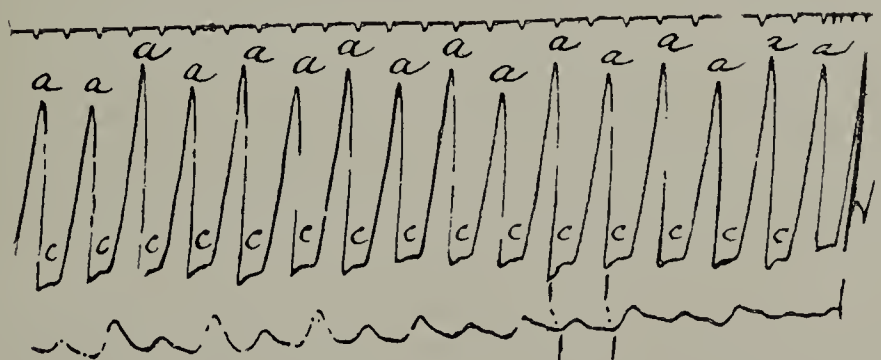


Fig. 38 (*Hcart*, 1909-10, I, 48). Polygraphic curve. Exaggeration of *a* waves during a paroxysm of auricular tachycardia, as a result of the coincidence of the auricular contractions and previous ventricular contractions. Synchronism of *a* and *v*. The *a-c* interval is .2 sec..

Supernumerary "a" waves.

If in a curve from a patient, where three clearly inscribed waves accompany each ventricular systole (waves which may be identified as *a*, *c* and *v*), a fourth wave occurs in the diastolic period of each cycle, it may be suspected that it results from an auricular systole which has given rise to no ventricular response. The interpretation is confirmed if the fourth wave is of similar form to the known *a* waves in the same curve, and if it is placed equidistantly between the preceding and succeeding *a* waves (Fig. 39). The pulse is generally slow. Where such a fourth wave lacks prominence and definition, and particularly where the remainder of the curve shows no evidence of damaged conduction in the heart, greater care must be exercised in the interpretation, for with relatively slow pulses a wave of different origin (first described by Hirschfelder³ and Gibson¹) is sometimes present. And it may happen that the wave (*b* or *h* as it has been called) falls equidistantly between *a* waves; when this difficulty is encountered, it may be overcome by observing the relationships of the several waves under several conditions of heart rate, occurring naturally, or induced by exertion, the suspension of respiration or the administration of drugs.

Changes in the frequency of the heart rate or the chance occurrence of longer pauses in the ventricular rhythm are also of value in the elucidation of instances of damaged conductivity, where the auricular and ventricular systoles fall together.

When in a venous curve the *c* waves and the points of termination of ventricular systoles can be clearly established, and there remains in addition to *c*'s and *v*'s a series of further waves, falling at regular intervals but bearing no fixed relationships to the ventricular systoles, the regular and added waves may be attributed to auricular systoles (Fig. 40). And in such instances it will be noticed that where an *a* wave falls during the limits of ventricular systole, its prominence is enhanced (Fig. 40).

Venous accompaniments of premature beats.

In the interpretation of curves in which single premature beats are occurring in the radial tracing, it is most essential to fix the points in the venous curve at which the ventricular systoles commence. And in such cases it may be necessary to calculate the transmission intervals for the premature beats themselves anew (Fig. 41). Having obtained the instants of their occurrence, search may be made for the missing auricular representatives; if an unexplained wave precedes the point arrived at, it may be taken as the representative of a premature auricular contraction, and

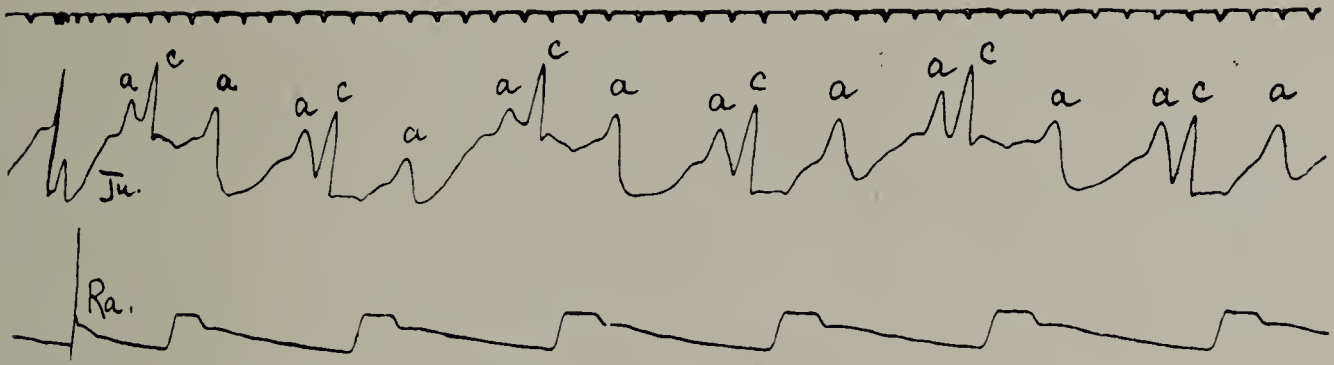


Fig. 39. Polygraphic curve. The *c* waves are each preceded by a presystolic wave *a*, and there are certain additional waves of similar appearance, falling outside the limits of ventricular systoles, and placed equidistantly to preceding and succeeding *a* waves. They are also the result of auricular contractions. The auricle is contracting at exactly double the rate of the ventricle.

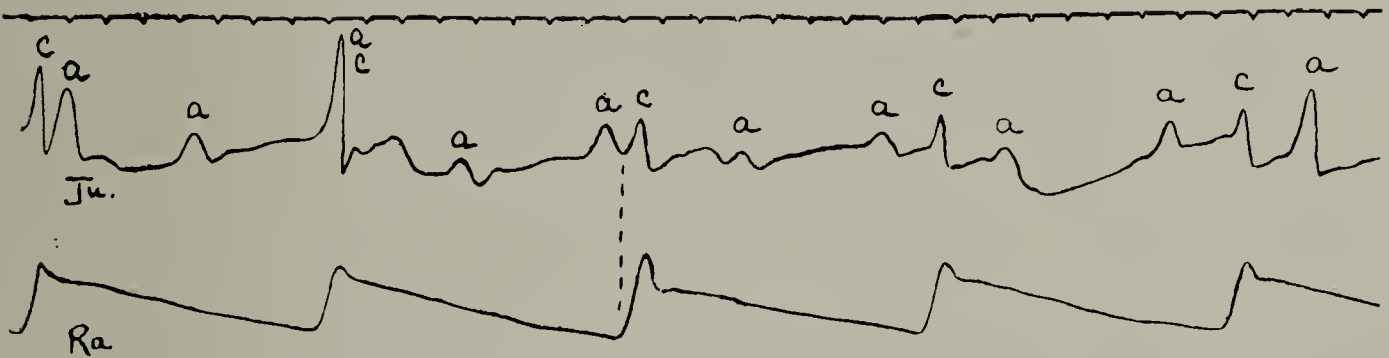


Fig. 40. Polygraphic curve. The *c* waves have been identified in the usual manner, and there remains a series of waves, scattered at approximately equal intervals in the curve, some of which are presystolic in time. They are all due to auricular contraction. Where, as happens in one instance, *a* and *c* fall together, an exaggerated wave results. The auricular and ventricular rhythms are dissociated; the auricle is beating somewhat more than twice as rapidly as the ventricle.

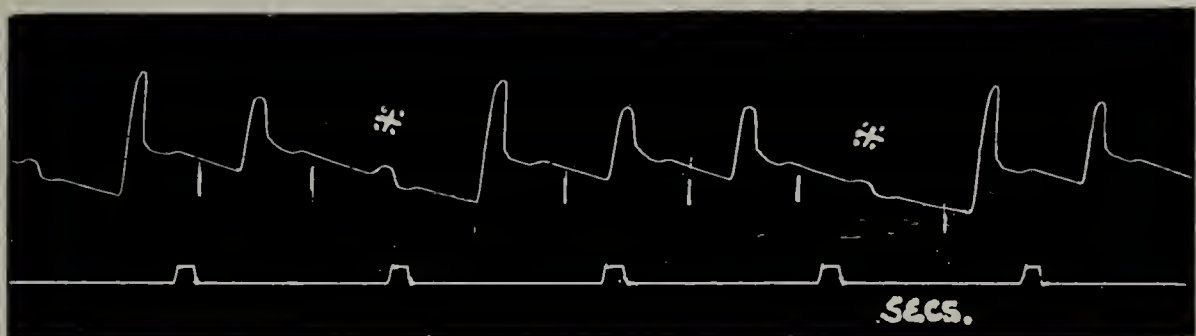


Fig. 41. A curve taken from a dog's carotid, by means of a Hürtle manometer. It shows two weak pulsations marked with asterisks. These resulted from premature contractions arising in the ventricle. Yet the carotid beats are not premature, but delayed. The increased delay may be calculated from the short ordinates which represent the actual times of onset of ventricular systoles, determined from a simultaneous curve taken direct from the ventricle. This possible fallacy in estimating the onset points of ventricular systoles from arterial curve has been pointed out by Hering.²

the interpretation is established if the premature beat disturbs the fundamental or dominant rhythm of the heart* (Fig. 42).

But if no such wave is found and an exaggerated peak occurs at the instant of ventricular systole, then the auricle and ventricle have contracted together (Fig. 35 and 36).



Fig. 42. Polygraphic tracing, in the radial curve of which a single premature beat occurs. It is accompanied by waves c' and v' . Each of the rhythmic beats is associated with a , c , and v waves. An additional wave (a') at the point marked with an asterisk is attributed to premature contraction of the auricle.

Absence of "a" waves from the jugular pulse.

Where the venous curve shows no sign of the presence of a waves, and when the chief peaks fall consistently within the limits of ventricular systole (the period E), the type of curve is spoken of by the name *ventricular form of venous pulse*. It occurs under several circumstances. The pulse may be regular or irregular. When regular the absence of true a wave is the result of:—

1. Distension of the auricle as an outcome of increased pressure in the chambers of the right side. An example of the type of curve obtained is given in Fig. 43. The corresponding electrocardiogram in which the representative of auricular contraction (P) is found is shown in Fig. 44.
2. Simultaneous contraction of auricle and ventricle, which may occur under several circumstances† (Fig. 33, 45 and 46).
3. Slow and regular action of the ventricle, associated with absence of co-ordinate contraction of the auricle (a condition fully considered in Chapter XIX).

* In measuring the pause, so as to ascertain the presence or absence of disturbance of the dominant rhythm, it is safest, for comparison, to take the beats which lie to the left of the disturbance in question, for the premature beat frequently disturbs the heart rate temporarily.

† Synchronous contraction may result from:—*a*). The auricular contractions falling back on preceding ventricular systoles. *b*). Retrograde contraction of the heart. *c*). The simultaneous response of auricle and ventricle to the same impulses. (Conditions more fully discussed in subsequent chapters.)

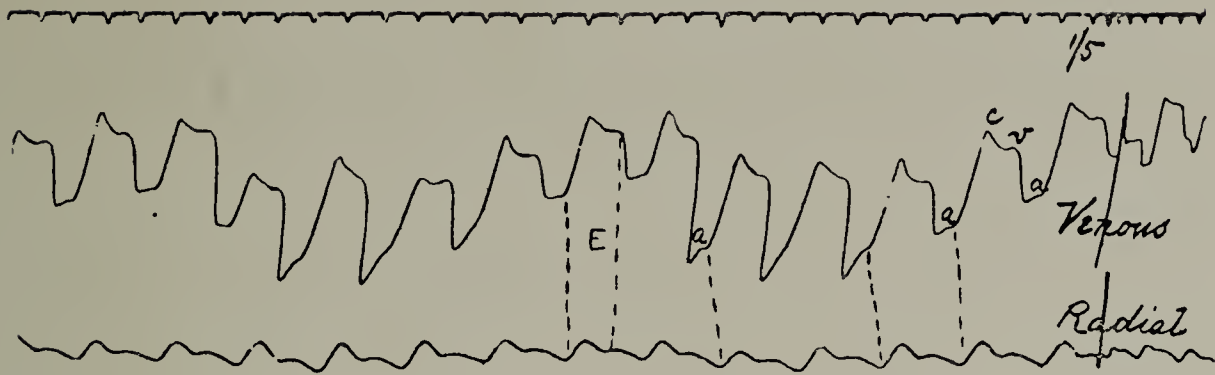


Fig. 43 (*Heart*, 1909-10, I, 358). Venous and radial curves, showing almost complete absence of *a* waves. The radial pulse is regular. From an instance of over-distension of the venous tributaries and engorgement of the right side of the heart.



Fig. 44. An electrocardiogram from the same case, showing a prominent *P* variation and proving the co-ordinate activity of the auricle.

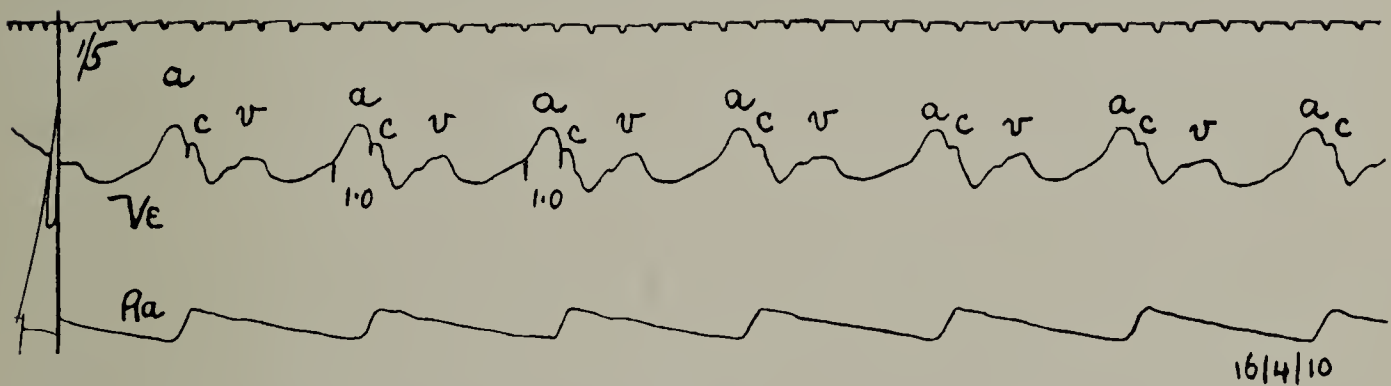


Fig. 45.

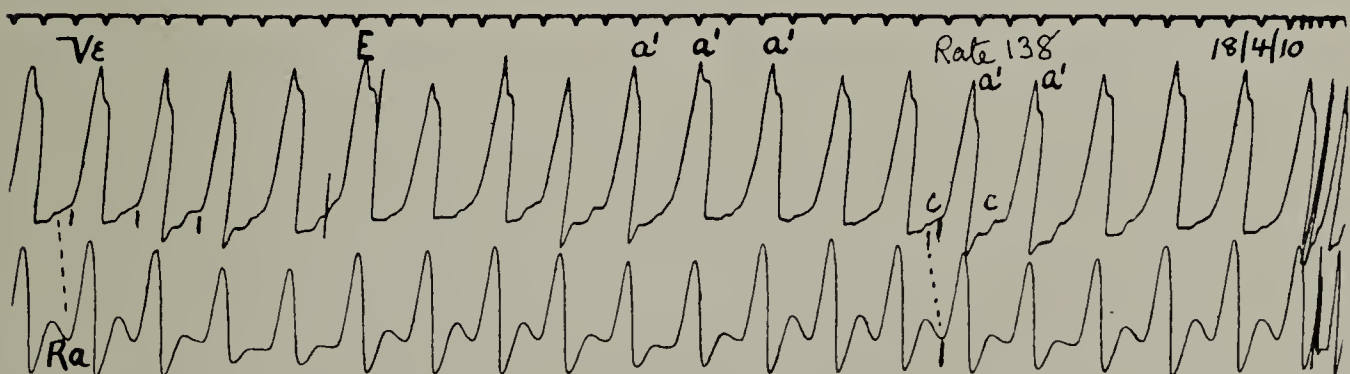


Fig. 46.

Fig. 45 and 46 (*Heart*, 1910-11, II, 130). Two polygraphic curves from a case of paroxysmal tachycardia. The first figure shows the normal curves of the slow periods. The second figure shows the ventricular form of venous pulse. The prominent waves fall during the period *E*, which marks the limits of ventricular systole. Resulting from simultaneous contraction of auricle and ventricle.

When the pulse is irregular the absence of true *a* wave is the result of inco-ordinate action of the auricles, a condition fully described in Chapter XVII.

Such are the general principles adopted in the analysis of phlebograms, principles outlined in the main by Mackenzie and established by a host of later workers.

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CHAPTER VII.

THE ANALYSIS OF THE CARDIAC MECHANISM. *ELECTRO CARDIOGRAPHIC CURVES.*

IN examining patients who are the subjects of disordered or irregular cardiac mechanism a certain preliminary knowledge of such mechanism is ordinarily, though not necessarily, acquired by one or other of the previously described methods ; the electrocardiographic investigation is usually undertaken as the most certain means of ascertaining or establishing the presence or absence of a suspected type of disorder. Thus the intervals between ventricular contractions may be accurately determined, and many of the general rules, which are of service in the examination of arterial curves, may be applied to the electric curves also. In these respects there is greater precision, and the electrocardiograms are more valuable, because beats of the ventricle, which are inconspicuous in the radial or apex tracing or which fail to affect arterial pressure, are clearly represented in them ; moreover the instants of onset of the contractions are accurately known ; there can be no error from miscalculation of transmission intervals. The instants at which auricular systoles occur may also be ascertained, and their relationships to the ventricular beats are readily discovered. The electrocardiogram, as a whole, presents a single and complete picture of the contractions of the upper and lower chambers, and the transference of points on one curve to another curve, necessitated by polygraphic work, is avoided. Nevertheless it sometimes happens, as we shall presently see, that certain of the auricular representatives in the electric curves are obscure and their recognition may be impossible. Under these circumstances, simultaneous venous and electric curves are of great service, for they often allow an accurate determination of the instants at which such auricular beats fall in the electric curve. Simultaneous electric and radial curves are of chief value in elucidating the arteriograms.

The recognition of auricular and ventricular contractions of physiological type.

Ventricular complexes which are of supraventricular origin.—When in leads from the right arm and left leg, the ventricular complex is of normal form, that is to say when it consists of two pronounced variations *R* and *T*, extending in the base-negative (or upward) direction, and when such

variations present the customary time relationships,* the systoles of the ventricle giving rise to them are known to be of supraventricular origin. They are due to contractions of the ventricle provoked by impulses descending along the normal channels, the auriculo-ventricular bundle and its arborisations, and reaching the ventricle through the normal field of reception.

The opposite conclusion, that an impulse entering the ventricle through the normal channels gives rise to a ventricular contraction which yields a complex of normal outline, is also true in most instances; and it probably holds good whenever a sufficient time has elapsed, after the preceding contraction, for the recovery of the tissue functions. It is a matter of indifference as to which area of the supraventricular musculature (the supraventricular musculature includes the whole of the right and left auricles, the auriculo-ventricular node and the bundle as far as its division) originates such an impulse. But, where the ventricular beat under consideration follows the preceding beat at a pronouncedly shortened interval, it occasionally happens that a more or less marked aberration may occur in the type of the electric curve to which it gives rise.^{7 & 8} The subject will be more closely examined in the chapter which deals with premature beats arising in the auricle. At the present time it will suffice to note that, despite the exceptions, the majority of ventricular beats called forth by supraventricular stimulus formation may be identified. The chief aberrant types of ventricular complex are known.

The identification of beats which arise in the vicinity of the pace-maker.—Given a ventricular complex of normal outline, the succeeding steps in the examination of the curve are directed to that portion of it which is presystolic in time; attention is fixed upon the auricular complex. If this segment of the curve presents a familiar outline, a rounded or peaked deflection in the direction of base-negativity (upward); or more especially if it is similar in its conformity to beats which are known to pertain to the regular and normally sequential rhythm for the heart of the patient investigated; then it may be asserted that the auricular contraction has its birth in an area of the right auricle in the immediate neighbourhood of the upper reaches of the sulcus terminalis, the customary if not invariable site of the pace-maker.

The identification of beats which belong to the normal rhythm proceeds along the lines laid down in the two preceding paragraphs. For except under special circumstances, hereafter considered, these heart beats will be accompanied by auricular and ventricular complexes of a usual and recognised form. And where incertitude remains, aid may be obtained from an examination of the arterial curves or from the accurate spacing

* R extending for a time distance of .02-.03 sec..

of such beats and a general consideration of the evidence acquired by the graphic methods as a whole.

The recognition of auricular and ventricular contractions of anomalous type.

The close dependence of the form of electric complexes, be they auricular or ventricular, upon the direction of contraction, and therefore upon the point or points of its initiation, has been previously emphasised. Examples of curve types deviating from the normal have been given (Chapter III), and others may be found in the chapter which deals with the electric curves accompanying premature contractions (Chapter XIII). Our acquaintanceship with the many and diverse types of electric curve is by no means complete at the present time, but many special forms of curve are known, and may be isolated as representative of contractions originating abnormally. Each example must be treated on its own merits; a full consideration of the facts, and the fixation of the time-relationship of the complex to the remaining complexes of the curve as a whole, is imperative. At present it is needful to lay stress upon the ascertained fact, that under special conditions a contraction which has started in normal fashion in the ventricle (*i.e.*, as the result of an impulse entering it through the normal field of reception) may give rise to a ventricular complex which has an outline closely resembling that which denotes a contraction of ectopic and ventricular origin* in another case (Fig. 47 and 48). The phenomenon enforces the need of the close comparison of anomalous complexes with the normal complexes of the same case.

The resemblance, between ventricular complexes associated with supraventricular impulse formation on the one hand and ventricular impulse formation on the other, gives rise to confusion chiefly in instances where the complex belonging to the supraventricular impulse is of aberrant type (Fig. 47 and 49). Now a deviation from the normal type of complex in an unhealthy heart is not necessarily attributable to an alteration of the onset point of the contraction; for although it is the rule that such deviation from the normal type is an indication of the alteration of the site of the *primum movens* in the ventricle, yet beyond doubt there are other factors which modify the form of the ventricular complexes. And no factor appears to be more potent in this respect than an alteration in the functions of the paths of conduction by which the impulse spreads. When an impulse enters the ventricular segment of the cardiac tube through the auriculo-ventricular bundle, it pursues its course to the point where the bundle divides and from this stage onwards is distributed through the medium of a complex system of branching conductors. Local damage to these paths must of necessity modify the form of resultant contractions, as it modifies the distribution of the discharge which awakens such contractions, and this expectation has been ratified by the experimental demonstration of Eppinger and Rothberger.²

* *I.e.*, a contraction arising at a single focus in the ventricle itself.

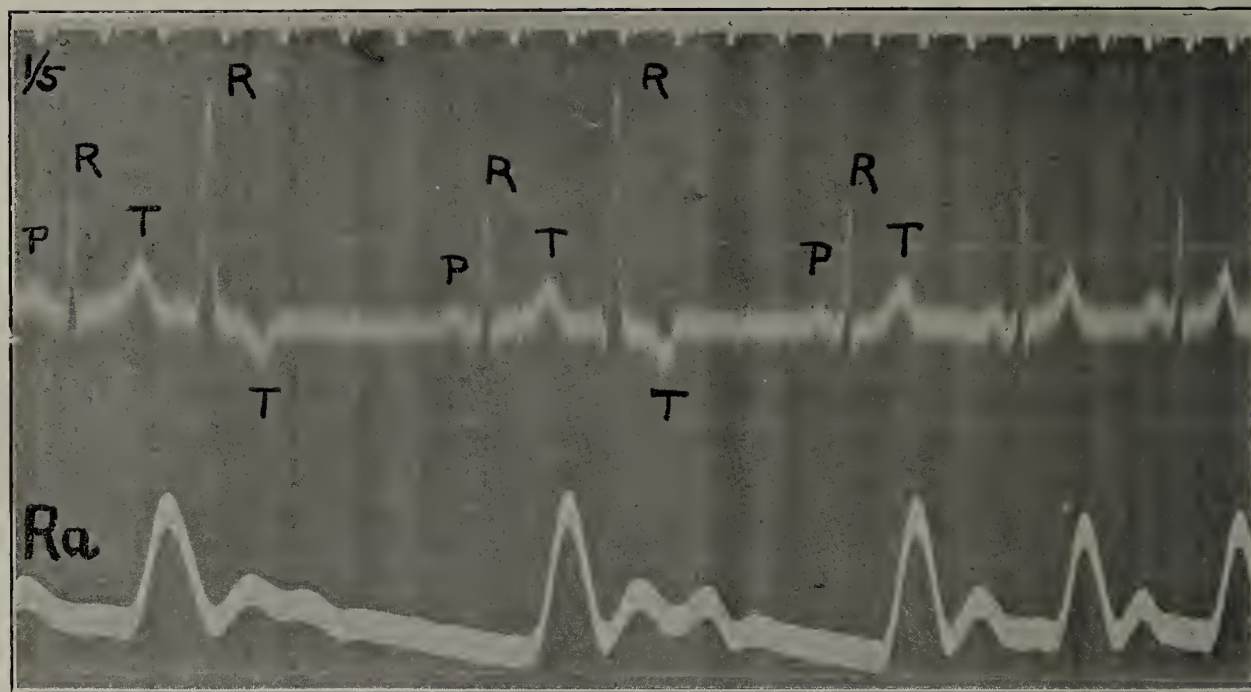


Fig. 47 (*Heart*, 1910-11, II, 23, *Fig. 11*). Electrocardiographic and radial curves from a patient exhibiting premature auricular contractions. Two such beats are shown, in which *R* is increased and *T* is inverted. These ventricular complexes are not dissimilar to those associated with premature contractions arising in the right and basal portions of the ventricle (*Fig. 48*); the two may be distinguished by noting the relatively short duration of the first (upward) phase. The auricular complex is not shown, it is represented by a horizontal effect.

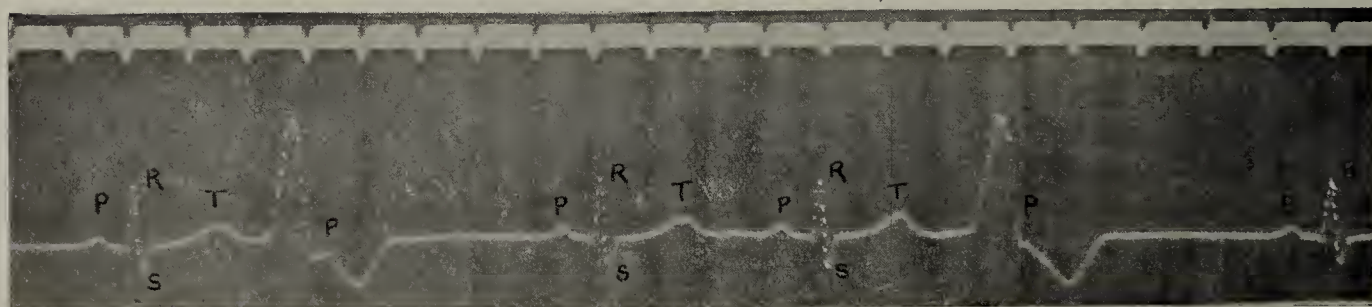
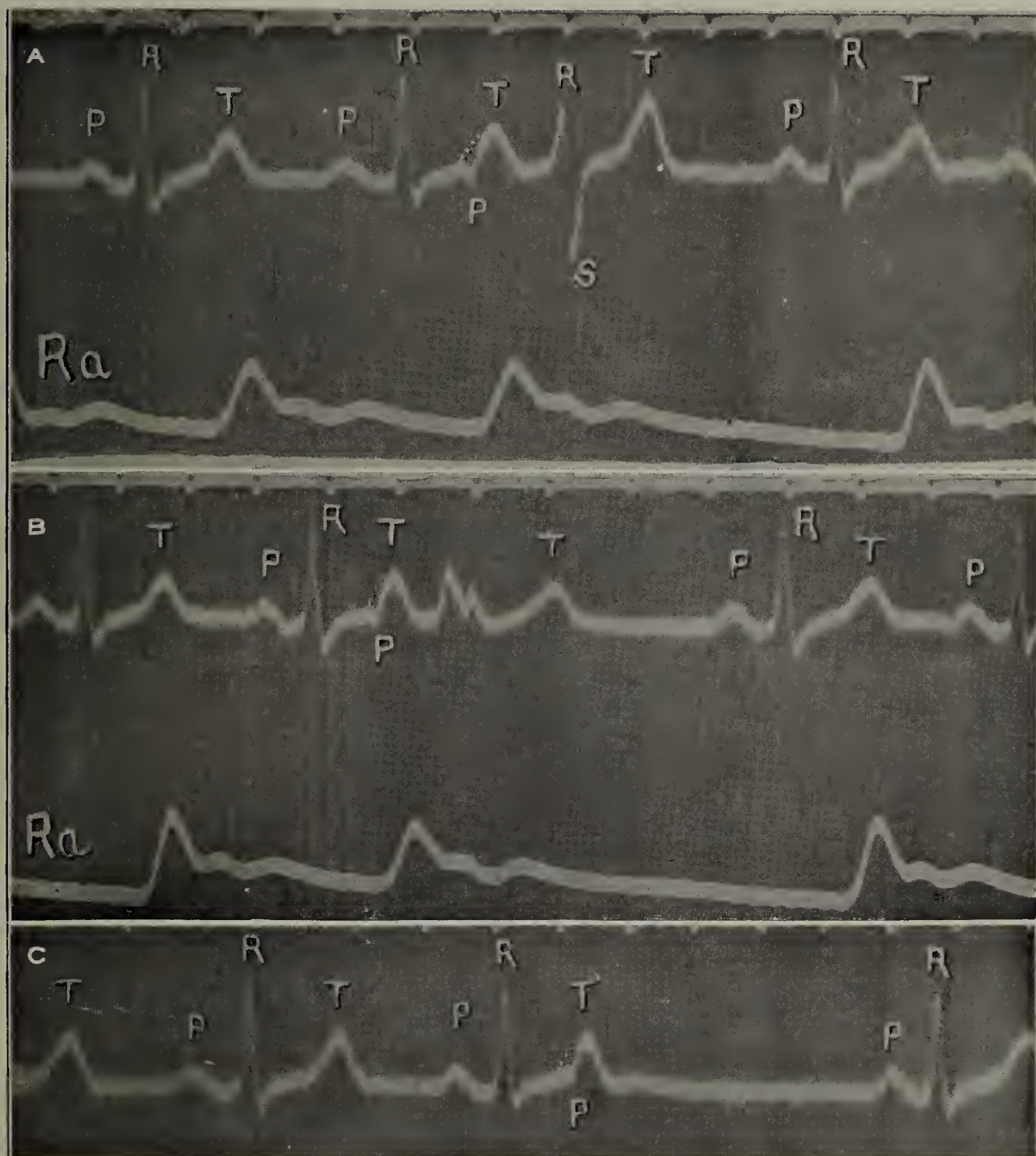


Fig. 48 ($\times \frac{2}{3}$). An electrocardiographic curve from a patient exhibiting premature ventricular contractions. Two such contractions are shown. They bear a superficial resemblance to the aberrant ventricular complexes of *Fig. 47*. The *P* summits of the regular auricular rhythm fall with these anomalous complexes.

Fig. 49 ($\times \frac{5}{6}$).

- A. Simultaneous electrocardiographic and radial curvo. The figure shows three complete heart cycles belonging to the normal rhythm, and one aberrant cycle, as a result of premature contraction of the auricle. The abnormal auricular complex falls upon the second *T* wave in the figure. Its shape is indicated by the dotted line; it is directed downwards and notches the *T* upon which it falls. It is followed by a ventricular complex, which though associated with a beat of supraventricular origin, is of aberrant form (*R*, *S* and *T*).
- B. A similar curve from the same patient. The events are similar in every respect, except that the ventricular complex which is premature is of a different type.
- C. From the same patient, showing a "blocked" premature auricular beat. It gives rise to no ventricular response.

The observations of Eppinger and Rothberger are of a very striking nature, and will have a far-reaching influence upon the study, not only of anomalous ventricular complexes, but of the normal electrocardiogram. Before reaching the region of the arborisations and papillary muscles, the normal impulse traverses the two main divisions of the bundle, as they lie upon one or other side of the intraventricular septum. In these situations they may be damaged experimentally and the resultant alterations of the ventricular complex are now known, thanks to the work of the authors referred to. Section of the left division of the bundle gives rise to a picture (Fig. 50 *A*) with which we are familiar, that yielded by stimulation of the right and basal portions of the ventricular musculature (Fig. 14*b*). Conversely, section of the right division, produces curves (Fig. 50 *B*) which closely resemble those yielded by stimulation of the left and apical portions of the musculature (Fig. 14*a*).

Now if damage to the system of conduction, where it is demonstrable, can change the type of the contraction yielded, it has to be allowed that modification of the ventricular curves may also arise when alterations of conduction occur either in the finer arborisations of the junctional tissues, in the propelling tissue itself (the main mass of the musculature), or in muscular bridges joining the separate layers of it. And as a result of such change, deviations of less conspicuous grade are to be anticipated not only from case to case, but also in one and the same case from time to time.

It may be thought for the moment that these possibilities seriously affect the value of the analysis of electrocardiographic curves, but such is not the case. For it is obvious from observation that the conditions under which they may be supposed to arise are of comparatively rare occurrence; and there is every reason to believe that with increase in our knowledge they will rather add to than detract from the information placed at our disposal.

Variations which are encountered in the outline of the electrocardiogram when the heart is regular and the normal sequence is present.

The electrocardiographic curves of heart beats, where the sequence of contraction in the chambers is normal and the rhythm regular, are of varied form from case to case, and modifications occur in one and the same case under different conditions. Thus the outline of individual curves alters as an accompaniment of changes of heart rate. It is also influenced by the administration of drugs which affect the heart. Our knowledge of such phenomena is still in an early stage of development; the variations from the normal as a result of the interference of these and similar factors are not profound, and they do not seriously affect the interpretation of the gross order of muscular contractions. In clinical cases, variations of a greater degree occur,

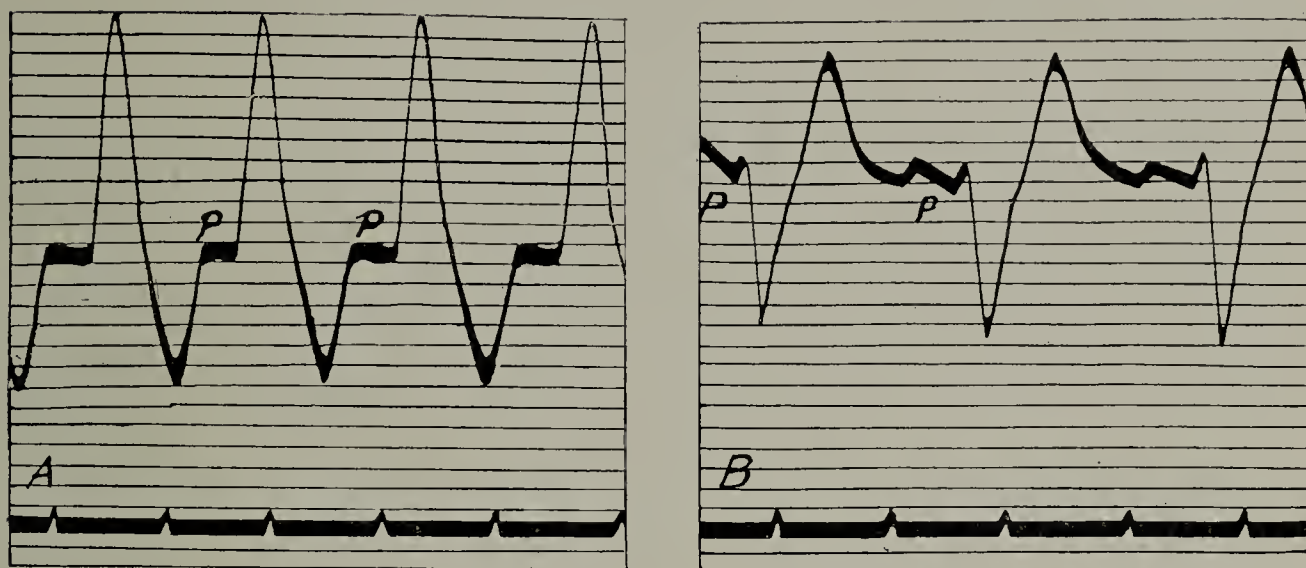


Fig. 50 ($\times \frac{2}{3}$) (after Eppinger and Rothberger, *Zeitschr. f. klin. Med.*, 1910, *lxx*, 5 and 11). Two electrocardiographic curves, resulting from section of (A) the left division and (B) the right division of the auriculo-ventricular bundle in the dog. Each ventricular complex is preceded by an auricular complex (P); the ventricular complexes themselves are of the form recognised as derived from intrinsic and anomalous ventricular contractions.

The observations of Eppinger and Rothberger are the more noteworthy, for Eppinger and Stoerk³ have happened upon clinical instances of the same phenomena and have been able to diagnose and subsequently demonstrate destruction of one division of the bundle at autopsy. Through the kindness of Professor Einthoven, the writer is able to reproduce a similar curve, from a patient, in whom, as we may now recognise, a lesion of the left division of the bundle was in all probability present (Fig. 51); a similar example, taken from a case under the observation of the writer, is shown in Fig. 52.

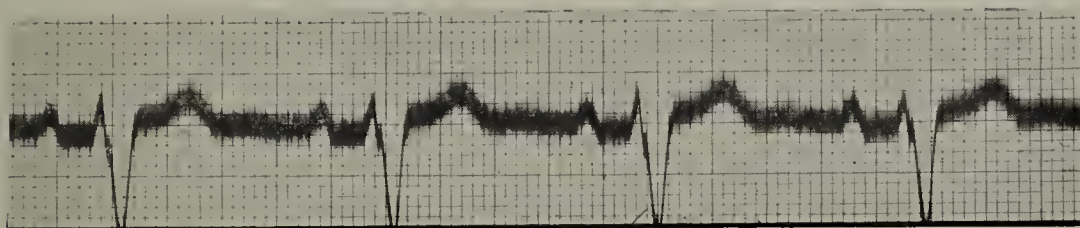


Fig. 51. A clinical electrocardiogram taken by Professor Einthoven. It shows well-marked P variations, each of which is followed by a ventricular complex of anomalous type and resembling that obtained on stimulation of the apical and left portions of the ventricle. Abscissæ = .04 sec.; ordinates = 10^{-4} volt. Probably the result of a lesion of the right branch of the bundle.

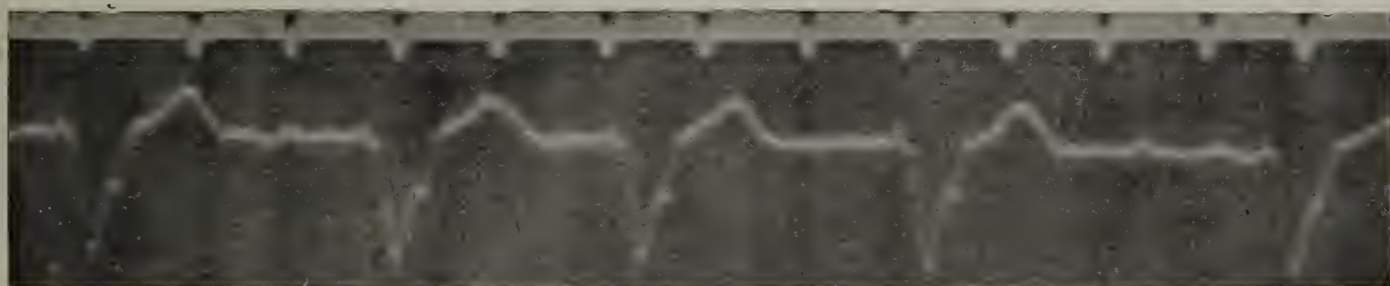


Fig. 52. A clinical electrocardiogram, showing similar ventricular complexes to those of Fig. 51. They resemble the curves obtained on excitation of the apex of the left ventricle (*cf.* Fig. 14). From a patient in which the auricle was not beating co-ordinately, and in which there was probably a lesion of the right branch of the bundle. Note the long duration of the first (downward) variation.

and chiefly as a result of morbid change in the heart wall and in the surrounding tissues. Here, again, the bulk of the facts have still to be collected, and we shall only deal with a few of the more definite instances of such variation, paying special attention to those, the recognition of which is of importance at the present time, either for their diagnostic value or because they have to be taken into account in judging of the points of origin of the contraction waves in the heart wall.

While the alterations in the type of the two complexes (auricular and ventricular) would in themselves offer sufficient material for a separate chapter, and while they should be recognised by the special worker, a detailed account of such changes would not materially assist the general reader at the present stage of the investigations directed towards their elucidation. We may confine ourselves to a consideration of two striking variations, one in the auricular, the other in the ventricular complex.

The auricular complex especially associated with hypertrophy of the auricle.—In cases of mitral stenosis, in which an increase in the bulk of the auricular musculature is so commonly encountered, the electric complex associated with contraction of the auricle is often so special in its form that it has been regarded as diagnostic of the condition. Whether it will be ultimately maintained as pathognomonic is a matter for further investigation, but it has been reported with sufficient constancy to render its consideration very important.^{1, 11 & 12} The auricular complex is inordinately large. Its amplitude may exceed one-fifth that of the peak *R* (in the physiological curve the amplitude is usually one-tenth that of *R*). It tends to have a flattened, rather than a rounded or pointed summit, and is often bifurcated (Fig. 53); it is prolonged.

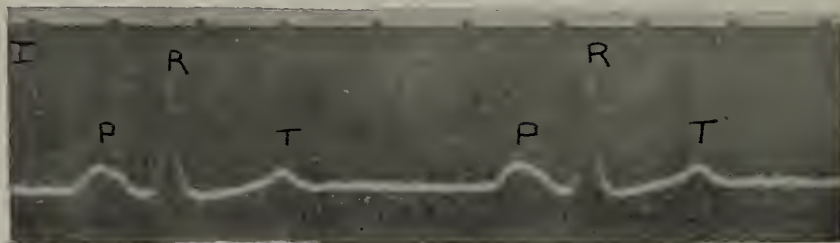


Fig. 53 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 127, *Fig. 9 I*). An electrocardiogram from a case of mitral stenosis, showing the increased prominence of the auricular complexes. It is broad and has a bifurcated summit.

Alterations in the magnitude and direction of the variation "T" of the ventricular complex.—Alterations of this nature have previously been referred to (Fig. 47), and are more fully dealt with in a later chapter, as accompanying premature beats of supraventricular origin. They are also



Fig. 54 (*Heart*, 1910-11, II, 23, *Fig. 13*). An electrocardiogram taken from a dog. Premature contractions were excited in the region of the sino-auricular node. Two are shown in the figure. Each is accompanied by an auricular and ventricular complex. The former is of normal outline (in the second cycle of the figure it is superimposed upon *T*). *T* varies according to the degree of prematurity. In the second cycle of the figure *T* is inverted. In the third it is exaggerated. Compare Fig. 55.

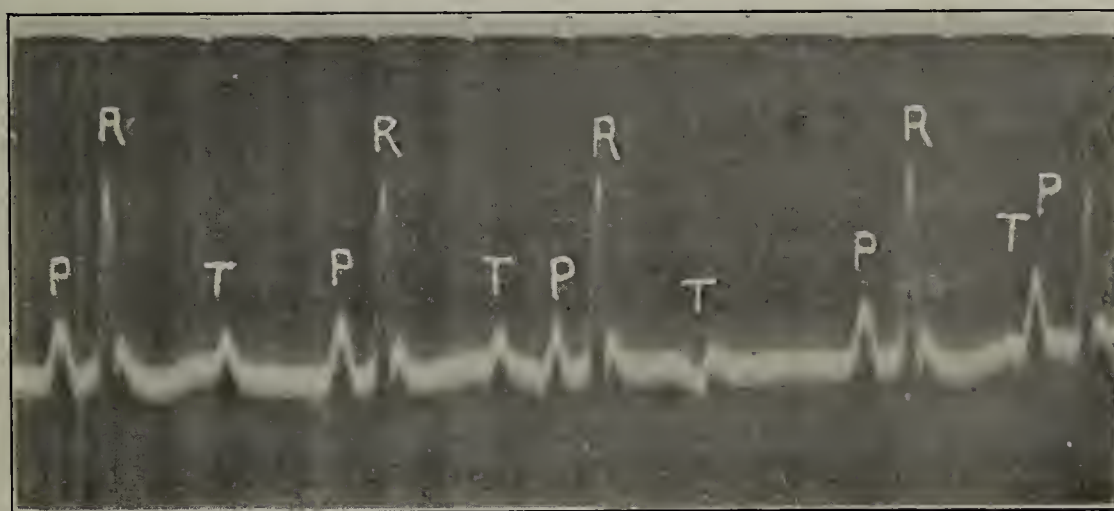


Fig. 55 (*Heart*, 1910-11, II, 23, *Fig. 14*). An electrocardiogram from the same animal. A premature contraction excited from the left appendix. The prematurity is less than in the first anomalous beat of Fig. 54, but greater than the prematurity of the second anomalous beat of the same figure. *T* is partially inverted. The anomalous auricular complex shows a small preliminary downward movement.

seen when the rhythm and sequence of the heart as a whole is normal. An exaggeration of T has been reported in association with exophthalmic goitre,⁴ it is not a constant accompaniment of this condition. It is of common occurrence in the experimental heart recovering from asphyxia (Fig. 56). It often appears to be related to marked increase in the energy with which the ventricular contents are expelled. Thus it is occasionally found in the first beat which follows the pause succeeding a premature contraction (Fig. 54 and 55).

But its nature is obscure and must remain so until we are more fully conversant with the meaning of the physiological electrocardiogram. Decrease in amplitude or actual inversion of T is more frequently encountered; and one might be led to suspect that it is related to deficiency in the energy of contractions, an explanation in harmony with that of its exaggeration. But the views of the mechanism of this modification are quite as problematic as those which attempt to aid us in understanding the exaggeration of T . Inversion of the wave is generally recorded from patients in whom there is reason to suspect grave myocardial affection, but is also encountered experimentally where no lesion exists (Fig. 56). Familiarity with the change is necessary, for the ventricular complex of which it forms a constituent part may bear a superficial resemblance to that which accompanies an anomalous ventricular contraction (Fig. 57 and 58). They may be usually differentiated by paying attention to the proportion between the total lengths of the first upwardly directed phase and the complex as a whole.

Those who are especially engaged in investigation, and desire further information in regard to variations in the form of ventricular complexes of ventricular beats, belonging to a regular and supraventricular rhythm, are referred to the recent publications of Pibram and Kahn,¹⁰ and Kraus and Nicolai.⁵

The algebraic summation of complexes.

In many examples of disorder of the cardiac mechanism, it happens that ventricular and auricular systoles fall synchronously; and in discussing the analysis of venous curves, we saw that simultaneous contraction of the upper and lower chambers is marked by the production of a special form of phlebogram, dependent upon the hindrance to the discharge of the auricular contents.

It seems that the electric complex of neither auricle nor ventricle is appreciably changed by such coincidence of their contractions, and the curve which results is consequently a simple composite of the complexes of auricle and ventricle. The auricular systole may fall at any point upon the ventricular systole, and its representative (P) may be found,

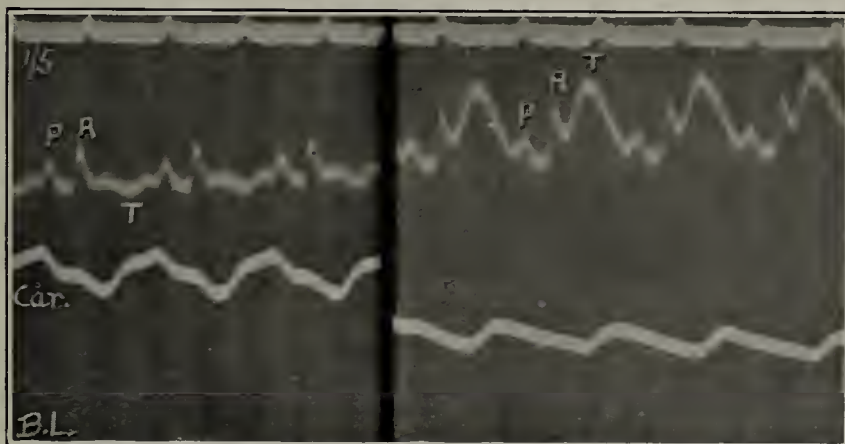


Fig. 56 ($\times \frac{2}{3}$). Electrocardiogram and Hürthle blood-pressure curve from a decapitated and curarised cat. The animal was asphyxiated for 4 mins. 30 secs.. The curve to the left of the stop was taken 15 secs., that to the right 30 secs., subsequent to the resumption of artificial respiration. To the right of the stop *T* shows an enormous exaggeration, to the left it is inverted. The simultaneous manometer curve shows a marked fall of blood-pressure with the exaggeration of *T*. The line, 0 mm. blood-pressure (*B.L.*), is represented by the lower border of the figure.

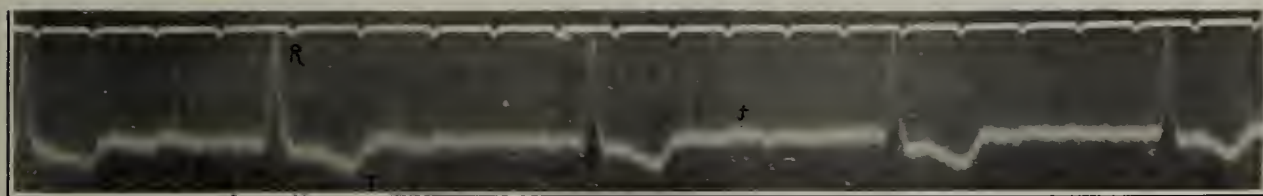


Fig. 57 ($\times \frac{5}{6}$). A clinical curve showing five ventricular complexes, in which *T* is inverted. The beats are all of supraventricular origin. (In this curve there is no evidence of co-ordinate auricular contraction: *cf.* Chapter XVII).

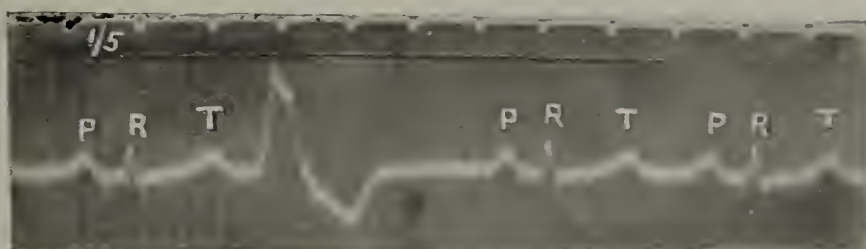


Fig. 58 ($\times \frac{1}{2}$). A curve from a patient the subject of premature contractions of ventricular origin. A single anomalous ventricular complex is shown. It bears a superficial resemblance to the curves of Fig. 57, but is distinguished from them by the longer duration of the first phase, the upwardly directed variation.

superimposed upon *R*, *S*, or *T*. The summation of effects is exact algebraically.

When complete dissociation of the auricular and ventricular rhythms is present, the *P* variations are of normal form and the *R*, *S* and *T* variations show a close resemblance to the normal type. The summation consists of a superimposition of auricular complexes, representing beats arising in the vicinity of the pace-maker, and of ventricular complexes having the general outline of beats of supraventricular origin¹ (Fig. 59).

When a premature ventricular contraction coincides with a rhythmic auricular systole, a *P* variation of normal type is superimposed upon an anomalous ventricular complex⁶ (Fig. 48 and 60).

It is not always easy to identify such auricular beats, for the exact and uncomplicated form of the anomalous ventricular complex may not be known. But where the prematurity of the ventricular contraction is variable, as in Fig. 48, the auricular portion of the curve is usually identified with ease by noting the differences in outline between the anomalous ventricular complexes. The auricular contraction when found is seen to occupy a midway position between preceding and succeeding auricular beats. Identification is also simplified when successive premature beats are encountered (Fig. 60); and in this instance, also, it depends upon a comparison of two anomalous complexes, and upon the spacing of the auricular contraction throughout the curves.

When a premature and ectopic auricular contraction* coincides with a rhythmic ventricular systole, an anomalous *P* variation is superimposed upon a ventricular complex of normal outline (Fig. 49).

In the preceding chapters we have dealt with the principles which guide the analysis of disorders of the cardiac mechanism and have spoken of the more important phenomena observed in studying arterial, venous, and electrocardiographic curves respectively. The observations outlined in these chapters will serve as a basis from which we may proceed to an examination of the chief disorders of the heart beat as they are met with in clinical work. Thus while the first section of this book deals in detail with the methods employed and the rules of interpretation, the second section will be devoted to a consideration of the disorders themselves and the correlation of these disturbances with experimental findings. It will be our endeavour to present the experimental and clinical facts side by side, and to set forth the proof of the identity of the naturally occurring disorder with that which may be produced by deliberate interference with the heart in the lower animals. At the same time the second section will contain the detailed evidence upon which many of the conclusions of the first section, and in particular the general rules of interpretation, are founded.

* A contraction arising at a point in the auricle removed from the pace-maker.

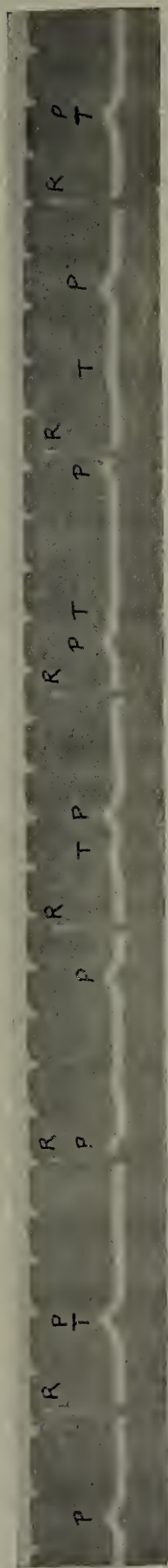


Fig. 59 ($\times \frac{1}{10}$). An electrocardiogram from a case of complete dissociation of the auricular and ventricular rhythms. Each ventricular beat is accompanied by *R* and *T* variations; the complex is of supraventricular type. Each auricular systole is represented by a *P* variation of the type known to proceed from the pace-maker. The *P* waves are scattered uniformly throughout the curve and fall in haphazard relationship to *R* and *T*, with which they summato.

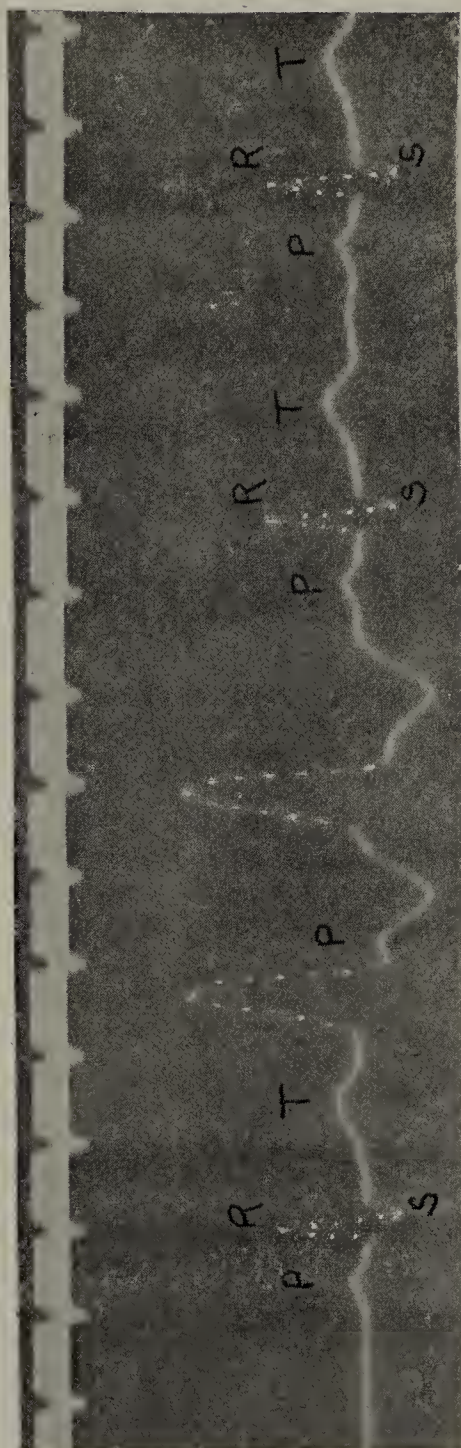


Fig. 60. A curve showing a pair of anomalous ventricular contractions. The sequential auricular contraction lies upon the downstroke of the first complex and midway between preceding and succeeding *P* waves. From the same case as Fig. 48.

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CHAPTER VIII.

EXPERIMENTAL HEART-BLOCK IN THE MAMMAL.

IN many organs in which a contraction is propagated as a wave transmitted in a direct line from point to point, transmission can be shown to depend upon the functional integrity of the tissues which directly unite adjacent sections of such organs. The experiments of Romanes upon the umbrella of the jelly fish, in which transmission of contraction first received careful study, laid the foundation of our present knowledge of the subject. His researches were succeeded by those of Gaskell, who worked with the amphibian and reptilian heart. The detailed observations of Gaskell clearly established the dependence of conduction upon direct continuity of the musculature, and gave us a clear conception of the mechanism of hindered conduction (heart-block) in the cold-blooded heart.

The final steps, the demonstration of similar phenomena in the mammalian heart, followed upon this work. Amongst the earliest experiments were those of Tigerstedt,²⁷ Wooldridge²⁸ and McWilliam²¹; these investigators were able to prove the subservience of the normal ventricular rhythm to impulses received from the preceding contractions of the auricle. We are chiefly concerned at the present time with the localisation of the path through which these impulses are conveyed in the mammalian heart, and the question is one of such supreme importance to clinical pathology that it seems desirable that the accumulated evidence should be set forth in detail. It is unnecessary to describe the earlier experiments referred to in the preceding paragraph, for excellent accounts of them have been already published; and the more important principles are equally well established by the work upon the mammalian heart. It will be sufficient if the reader is referred to Romanes' book, *Jelly Fish, Star Fish and Sea Urchins*, published in the International Scientific Series, and to Gaskell's article in the second volume of Schäfer's *Text Book of Physiology*.

The evidence that the auriculo-ventricular bundle transmits the impulses from auricle to ventricle in the mammalian heart.

The fact that dissociation of the usual co-ordinate sequence of contraction in the chambers of the mammalian heart may occur, has been known for many centuries, though the cause of it remained obscure for a long while. Harvey, in his essay on the movements of the heart, stated

that he noticed a prolongation of the interval between the auricular and ventricular systole, and observed an occasional ventricular contraction following upon several auricular contractions under similar circumstances. The earliest attempts to dissociate the auricle and ventricle, made by Tigerstedt and Wooldridge, have little more than a historic value from our present point of view. They were successful, but involved widespread damage to the heart. Two years after the publication in which His described the auriculo-ventricular bundle, the same observer^{12 & 13} attempted, in conjunction with Graupner, to destroy the connection between auricle and ventricle in the heart of a rabbit, and apparently with success. In a preliminary communication he records that dissociation of the auricular and ventricular rhythm was produced (1895). No tracings of the heart's mechanism and no histological evidence of the lesion were published.

Humblet¹⁴ (1904) operated upon the heart *in situ*. Having tied the vena azygos and obstructed the inferior vena cava and the superior vena cava, he opened the right auricle and made lesions in the region of the bundle, subsequently closing the wound in the auricle and restoring the circulation. The method proved unsuccessful in his hands, and he discarded it in favour of that of perfusion of the isolated heart. Dogs were employed. He reported that cuts in the neighbourhood of the bundle were without effect, but that when the bundle was damaged dissociation appeared. There is no mention of subsequent histological examination of the bundles, and the single tracing which is given is unsatisfactory.

At a later date Humblet¹⁵ undertook further observations upon the dog's heart. The ventricle of the excised heart was nourished by transfusion; ligatures were passed around the bundle and drawn tight. Eight experiments yielded seven definite results; in each case dissociation was obtained. Curves illustrating the abnormal mechanism were given from three of the animals and also drawings of the lesions as they appeared microscopically. In the eighth experiment heart-block appeared spontaneously.

Hering's results⁹⁻¹¹ were published in 1905-6. The bundle was damaged in the heart of four dogs. In three instances in which its tissue was entirely transected complete heart-block occurred (curves from two of these animals have been published). In the fourth animal the bundle was only partially divided, and in this instance it is stated that no heart-block manifested itself. The histological examinations were made by Tawara.²⁶ Hering also demonstrated that lesions breaking the anatomical continuity of auricle and ventricle at points other than that at which the bundle is found do not lead to dissociation.

In 1906 Erlanger⁶ published his first observations; attempts were made to pass ligatures through the heart beating *in situ*, in such a way as to ensnare the bundle. In seven experiments heart-block was seen once. The method was abandoned. In a second series of experiments an auriculo-ventricular clamp was utilised. Seven experiments yielded

heart-block on two occasions. Eventually he employed a special clamp, the lower blade of which consisted of a long-shafted fish-hook bent at a right angle. Introduced between aorta and pulmonary artery, the point of the fish-hook was carried into the left ventricle and thence through the ventricular septum below the bundle. The blades of the clamp could be tightened to a desired extent, and the bundle crushed. In seventeen experiments upon the heart of the dog beating *in situ*, heart-block was obtained in sixteen instances, and these, according to the histological findings of Retzer, were the experiments in which the bundle had been damaged. The observations have been extended and confirmed by this worker, and his collaborators^{7 & 8} and also by Tabora,²⁵ using a similar method. Biggs¹ (1908) stated that section of the bundle gives complete dissociation in rabbits; but no confirmatory histological observations were made.

The latest work is that of Cohn and Trendelenburg.³ The observations were conducted upon the perfused heart, and the work is conspicuous for the number of experiments and the care with which they were performed and reported. In all, the hearts of 26 cats, 17 dogs, 4 rabbits, 2 apes and 4 goats were successfully investigated. In each instance a complete account of the resulting disturbance in the heart has been reported, together with a diagram of the bundle and lesion, reconstructed from the examination of serial sections; usually the protocol is accompanied by an actual tracing of the heart's mechanism, frequently by macroscopic photographs of the lesions themselves. The results of this work, remarkable for the thoroughness of its execution, may be stated in general terms. Where the incisions failed to reach the bundle, or where it was but partially transected, no disturbance, or a partial or temporary heart-block, was encountered. Where the bundle was completely transected, complete heart-block invariably ensued.

The experiments so far considered all lead to the conclusion that the auriculo-ventricular bundle is the essential organ of conduction; its destruction yields a uniform result, complete dissociation of the auricular and ventricular rhythms; while damage to the surrounding structures or to the remaining anatomical connections between one and other chamber are without effect.^{3, 6, 11 & 15}

Certain observations from the Berne school have been at first sight contradictory, and these may be considered in further detail.

Kronecker and Busch¹⁸ (1899) worked with *rabbits*, and stated that the bundle might be broken without interference with the heart's sequential rhythm. The histological proof of transection is wanting. Kronecker¹⁹ himself reported similar results in the dog. Imchanitsky¹⁶ (1906-7) used the rabbit's and dog's heart, the organs beating *in situ*, and worked by the method of ligation adopted by Kronecker. In the instance of a rabbit

in which no dissociation was obtained, it is stated that the microscopic examination showed destruction of the bundle. It is not stated that serial sections were cut.

Lastly, Paukul²² (1908) reports the general results of experiments upon 24 rabbits; he followed Kronecker's method. He concludes that when the bundle is caught in the ligature without much damage to the surrounding structures, no dissociation occurs; and that dissociation may occur when the ligature passes in the neighbourhood of the bundle, leaving it intact. He gives histological drawings and the curves from several animals as evidence of these assertions.

The following objections may be urged against the conclusions of the Berne workers. In the work of Kronecker and Busch and Kronecker the histological proof of the bundle transection was not given. The solitary observation of Imehanitsky and the more extensive observations of Paukul, which alone demand serious attention, are accompanied by a report of the histology. But the sections were cut at right angles to the course of the bundle, and it is probable that branches of the bundle leaving the main tract in the earlier part of its course would escape observation as a result of the employment of this technique. The argument becomes more weighty when it is known that such early outgoing fibres are usually present in the rabbit.³

The experimental work upon the auriculo-ventricular bundle has been given in full, not only because a conclusion which is fundamental to cardiac pathology rests upon it, but because the review allows a limited presentation of the experimental methods adopted in such heart work, and further because it impresses the necessity of bearing clearly in mind the species of animal upon which such researches have been executed.

In summing up the observations, it may be said that in regard to the work upon the dog no doubt remains, and in regard to the experiments upon other animals, the results are concordant with the single exception of the rabbit.

Treated by themselves, the reports of observations upon this animal alone might lead to hesitancy in the formation of a definite conclusion as to the means by which the transmission of impulses from auricle to ventricle is affected. Considered in conjunction with the overwhelming testimony of experiments upon other species, a definite conclusion may be formed, which is strengthened by the knowledge of the special arrangement of the bundle branches in the rabbit. It may be held as demonstrated that a limited tract of tissue stretches between auricle and ventricle, and that the co-ordination of auricle and ventricle is dependent upon its integrity. Further, it may be stated that the integrity of the anatomical union between auricle and ventricle at all other points is immaterial to impulse transmission.

In the clamp experiments of Erlanger and in the section experiments of Cohn and Trendelenburg, it not infrequently happened that when the

damage to the bundle was limited the grade of heart-block showed a parallel variation. Thus, in Erlanger's experiments, successive turns of the screw tightening the clamp produced increasing degrees of heart-block, and this investigator obtained results which stand as a complete confirmation of the clamp experiments of Gaskell upon the tortoise heart.

By compression of the bundle all stages of heart-block were obtained and these included :—

- a) An increase in the interval between the onset of auricular and ventricular systoles ; an increase of the *As-Vs* interval as it is termed, which in the normal dog approaches .1 sec..
- b) An occasional failure of the usual ventricular response to the regular auricular impulses.
- c) Regularly occurring ventricular silences ; the failure of response of the ventricle to each tenth, ninth, eighth, seventh, sixth, fifth, fourth or third auricular contraction.
- d) A failure of the ventricle in its response to alternate auricular beats ; the establishment of a so-called 2 : 1 rhythm, wherein auricle beats at precisely twice the rate of the ventricle.
- e) The response of the ventricle to each third beat of the auricle ; the so-called 3 : 1 rhythm.
- f) The onset of a complete dissociation between auricular and ventricular rhythm, *i.e.*, the entire failure of transmission of auricular impulses, coincident with the awakening of the dormant rhythm inherent in the ventricle.

Such disturbances of the rhythm of the heart are the usual outcome of injury to the auriculo-ventricular bundle, as it stretches from the node of Tawara to the point where it divides. They do not follow upon section of one or other branch subsequent to the division.³ They do not necessarily follow when the bundle is partially divided ; it is perfectly apparent from the experimental findings that conduction may be unimpaired when a partial section is executed, and the same experimental findings definitely suggest that where such a partial section occurs and complete dissociation supervenes, it is the result of section of a part and damage to the remainder of the conducting tissue. A portion of the bundle with its two intact branches, or the whole bundle with one or other of its branches, is sufficient, if undamaged, to preserve an efficiently conducting tract.

Erlanger's experiments⁸ upon dogs have shown that a bundle when once destroyed is not repaired. He succeeded in restoring animals in which the connecting bridge had been broken, and in those animals in which the section was shown to be complete, the dissociation was full and the result permanent.

Such are the main conclusions which may be drawn from the experiments in which the auriculo-ventricular bundle has been injured.

Heart-block as a result of other interferences.

But it may be asked whether heart-block, partial or complete, is of necessity the outcome of a destruction of tissue; the reply to this question is definitely in the negative.

Heart-block may occur as a simple result of stimulation of the vagus nerve,² and heart-block of this nature will be more fully considered in later chapters.

It may also be brought about by the injection of poisons, and notably by digitalis,^{4 & 25} by aconitine,⁵ by adrenalin,¹⁷ and by muscarine and physostigmine.²³ It also results directly from asphyxia.^{20 & 24} In asphyxia it may be shown to be independent of blood-pressure, of vagal inhibition, of excess of CO₂ and of dilatation, and it probably owes its onset to lack of oxygen, perhaps to the resultant accumulation of acid products in the cardiac musculature (Mathison). The heart-block which ensues in cardiac poisoning may be of any grade, partial or complete (Fig. 61-63), and there may be, and often is, complete and rapid recovery from the derangement of function with the removal of the cause (Fig. 64). Thus in asphyxia, recovery from heart-block, whether partial or complete in grade, is the rule at the reestablishment of ventilation.

Fig. 61 ($\times \frac{1}{2}$) (*Lewis and Mathison, Heart, 1910-11, II, 47, Fig. 1*). Four electrocardiograms from a single period of asphyxia; taken from a decapitated and atropinised cat. They show varying grades of heart-block in the order of their onset. The *P-R* intervals are marked in seconds.

- I. Curve taken 1 min. after the onset of asphyxia. Auricular contractions are represented by waves *P*; ventricular systoles by waves *R* and *T*. The *P-R* interval is normal, .08 sec..
- II. Taken 5 min. 30 sec. after the onset. The *T* wave has become inverted. The *P-R* interval has increased to .13 sec.. It first showed increase at 4 min. 15 sec..
- III. Taken 6 min. after the onset. 2:1 heart-block is present. Alternate auricular contractions fail to give ventricular responses.
- IV. Taken 7 min. 30 sec. after the onset. Complete dissociation is present. Two ventricular complexes are shown; the first *P* falls in the centre of a ventricular contraction, the second falls clear, and the third coincides with the summit of *T* of the second ventricular complex. Recovery from this condition was complete.

Fig. 62 ($\times \frac{2}{3}$) (*Lewis and Mathison, Heart, 1910-11, II, 47, Fig. 2*). A curve taken from a decapitated cat soon after the onset of asphyxia. It shows three dropped beats, the lengthening of the *P-R* interval which leads up to them and the shortening which succeeds them. Certain of the *P* variations fall back upon preceding ventricular complexes. The *P-R* intervals are marked in seconds.

Fig. 63 ($\times \frac{2}{3}$) (*Heart, 1910-11, II, 47, Fig. 3*). A curve taken from an intact cat 3 min. after the onset of asphyxia. Showing dropped beats and lengthening of the *P-R* interval.

Fig. 64 (*Heart, 1910-11, II, 47, Fig. 4a*). From the same animal, showing the rapid recovery from 2:1 heart-block upon the cessation of asphyxia.

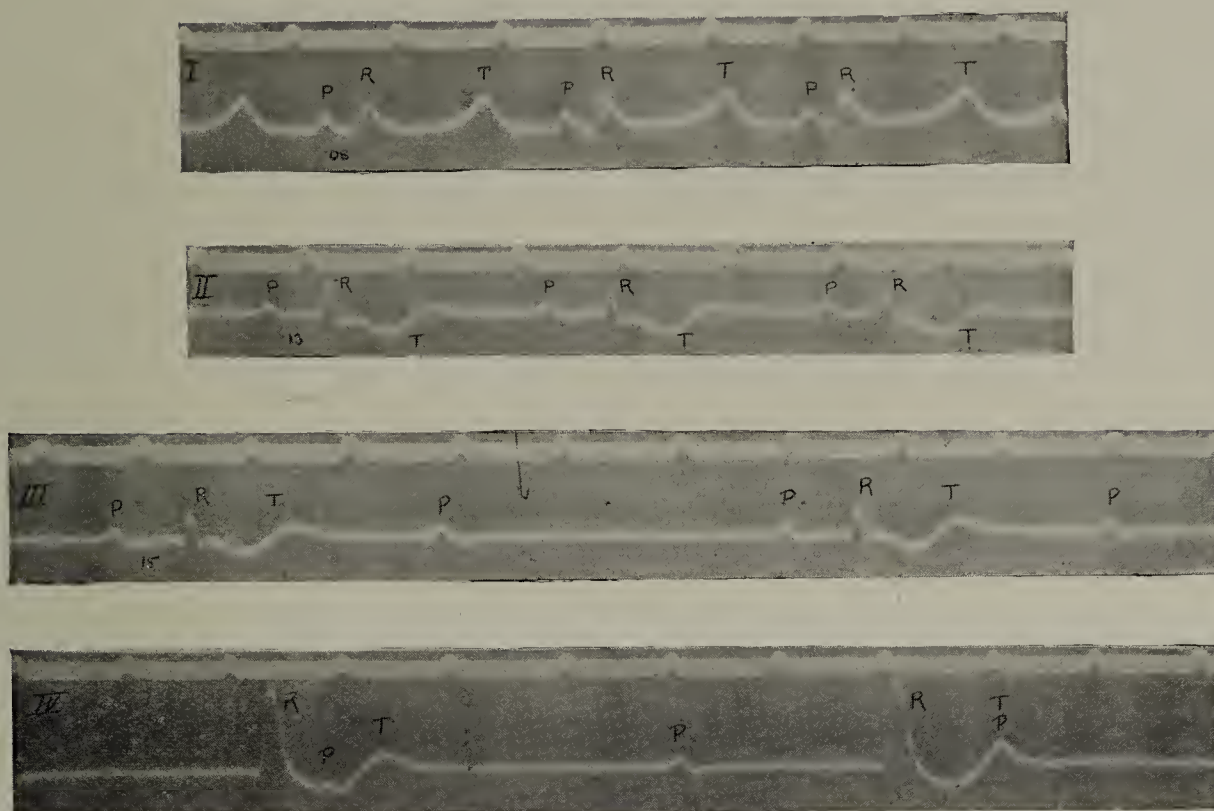


Fig. 61.

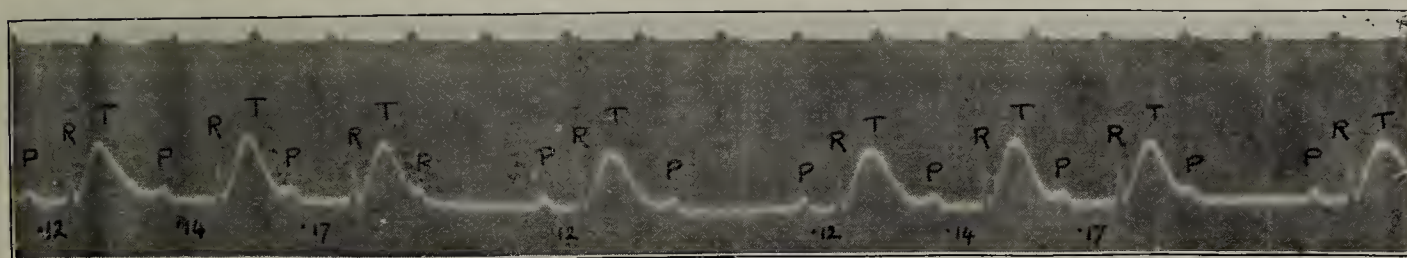


Fig. 62.

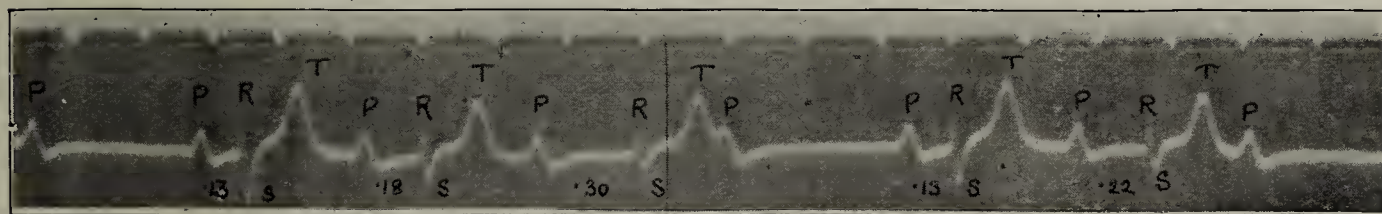


Fig. 63.

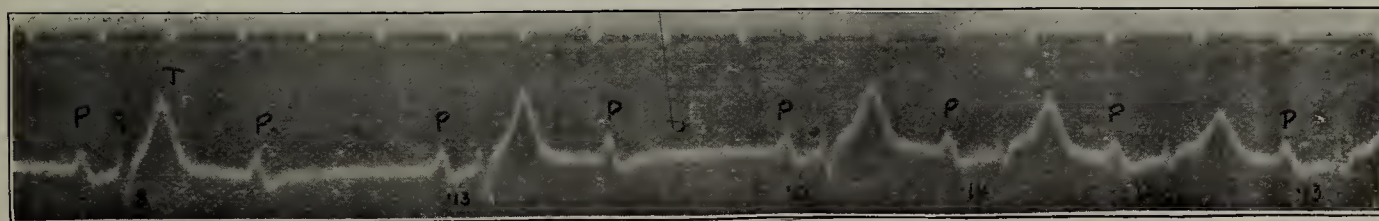


Fig. 64.

*Records of experimental heart-block.**

Records of the mechanism of the heart beat, subsequent to the damage of the *A-V* bundle, or subsequent to other interferences productive of heart-block, may be taken in many ways; but we may confine ourselves at the present time to a brief description of the electric curves, for they are peculiarly legible, and are demonstrative in every way. The curves obtained during asphyxia are characteristic examples of the mechanism.

If a cat is asphyxiated for periods varying from 1-7 minutes, a regular succession of events is observed in the heart. Within 1-3 minutes of the onset of asphyxia, the *P-R* interval (representative of the *As-Vs* interval) shows a notable prolongation (Fig. 61, *I* and *II*). This phase may last for a shorter or longer time, and is succeeded by a condition in which single responses of the ventricle to auricle are missed (Fig. 62 and 63). At this stage the *P-R* interval shows changes of great interest and importance; they are illustrated by Fig. 62 and 63. The *P-R* interval, which is primarily increased, demonstrates a gradual and further increase up to the point where the response is missed; and the eventual prolongation may be so great that a given auricular representative falls back, and coincides with a previous ventricular contraction (the *P-R* interval in experiment may lengthen to .3 sec. at least). Subsequent to the failure of transmission and following the resultant ventricular pause, the interval decreases abruptly and the whole process is repeated. The alteration of the *P-R* interval changes the time relationships of the ventricular beats. The dropping out of a single ventricular beat is not accompanied by a pause equal to two ventricular cycles, but to a pause of shorter duration. Thus the grade of the irregularity in the ventricle is diminished. Minor changes in position occur in the remainder of the ventricular cycles; as the *P-R* interval increases, the ventricular beats may quicken or slow respectively, according to whether the rate of increase in *P-R* interval diminishes or accelerates.

The phase of prolonged *P-R* interval with dropped beats is succeeded by one of 2:1 rhythm, in which each alternate systole of the regular auricular rhythm awakens a ventricular response (Fig. 61, *III*). Finally, the mechanism passes to one of complete dissociation, in which independent rhythms are found in auricle and ventricle, each regular, but bearing no simple mathematical ratio to each other (Fig. 61, *IV*). In the electric curves representatives of ventricular contraction (*R* and *T*) are found at regular intervals in the curve; and also representatives of auricular contraction (*P*), scattered uniformly through the curve, but falling in haphazard relationship to the ventricular contractions. Where auricular and ventricular contractions coincide, accurate summation of the electric effects are noted.

* While the experimental records of mammalian heart-block are described before the clinical for purposes of convenience, it is interesting to note that the majority of the observations were first obtained upon patients.

With the re-establishment of artificial respiration, recovery ensues (Fig. 64). It is extremely rapid and may follow any grade of heart-block.

The relationships of the experimental work to clinical observation will be discussed in succeeding chapters. It may be well to bear in mind that experimental heart-block is of two forms. In the one case it results from a localised lesion; in the other instance it follows an interference (toxic or innervatory) in which a large area of the cardiac muscle is involved. The second class of experiment suggests that the junctional tissues have a peculiar functional susceptibility, and we shall notice a striking parallel to the experimental findings when we turn our attention to the therapeutic action of certain cardiac drugs.

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CHAPTER IX.

CLINICAL HEART-BLOCK.

THE first suggestion of dissociation of the auricular and ventricular rhythms in man dates back to 1875, when Galabin³ reported a case of slow ventricular action (25-30 per minute) and remarked, "we have here a heart, the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long pause instead of just before the systole of the ventricle." He based his account upon the auscultatory phenomena, and upon curves taken from the heart's apex. Two excellent tracings which he published show beyond question that he was dealing with what we now recognise as complete heart-block. And these observations are the more noteworthy because they were published in the same year as those of Romanes in the *Philosophical Transactions*, and several years before the work upon the cold-blooded heart had been accomplished.

In 1885, Chauveau¹ described a case of heart affection and was able to recognise, in tracings taken from the radial pulse, the apex beat and the neck, that auricle and ventricle were beating at different and entirely independent rates. He compared the phenomenon to the dissociation obtained upon stimulation of the vagus.

In the year 1899, Wenckebach⁹ and His⁵ both described heart-block in the human subject and suggested a lesion of the auriculo-ventricular bundle as its cause. Wenckebach based his diagnosis entirely upon the arrhythmia produced in the ventricle. His gave polygraphic curves. The early publications of Mackenzie^{7 & 8} notably advanced our knowledge of the subject, and during the last few years a multitude of cases have been placed on record.¹⁴

The signs of heart-block in man.

The evidence of the occurrence of clinical heart-block is of the most definite character; it consists in the main of observations recorded graphically, and of a comparison between these records and those obtained in experiment.

In certain cases in which the pulse is slow, the fact that auricle and ventricle are contracting at distinct rates may be observed and recorded. A rapid movement at the base of the heart and in the situation of the right auricle has been described by several of those who have examined their cases radiographically.¹³

The radial pulse is slow and the beats are generally forcible ; systolic pressure is high, and during the long diastolic pauses which follow each pulse wave there is a considerable drop in pressure. Not uncommonly a faint and as yet imperfectly understood indication of the auricular systole is found upon the radial tracing¹¹ (Fig. 65).

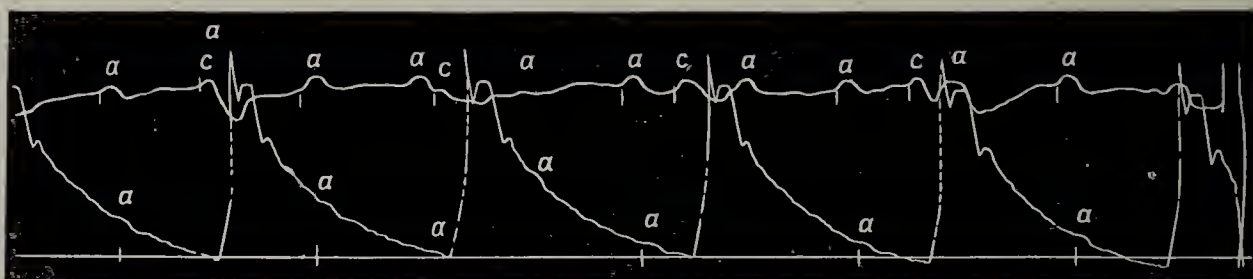


Fig. 65 (a retraced Dudgeon curve). A polygraphic curve from a case of heart-block, showing *a* waves on both jugular and radial curves.

At the apex beat there is no difficulty in recognising the ventricular beats, and auscultation may reveal the presence of dull and distant sounds, which are attributed to auricular movements.¹² Many of the apex curves show unmistakable evidences of the same events. Curves taken from the epigastrium present similar features.

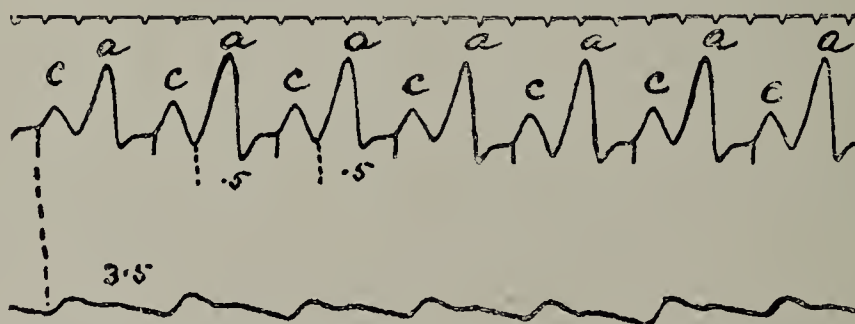


Fig. 66 (*Quart. Journ. Med.*, 1908-9, II, 359). Venous and radial curves from a case of mitral stenosis under the influence of digitalis, showing great prolongations of the *a-c* interval. It amounts to .5 sec. as opposed to the normal interval, .2 sec.. From the same case as Fig. 32 and 36 etc..

The venous curves are of great value. With each ventricular systole *c* and *v* waves appear, and in addition waves (*a*) resulting from the systole of the right auricle are found scattered at uniform and frequent intervals in the curves ; they may be twice, thrice or four or more times as numerous as the representatives of the ventricle. In partial heart-block, each ventricular systole is preceded by an *a* wave (Fig. 66-69). In complete heart-block the *a* waves fall regularly but with haphazard time relations to the ventricular representatives (Fig. 70).



Fig. 67 (*Quart. Journ. Med.*, 1908-9, II, 361). A curve from the same patient as Fig. 66, showing intervals ranging from .3 to .6 sec.. One beat, the expected response to a^9 , has dropped out.

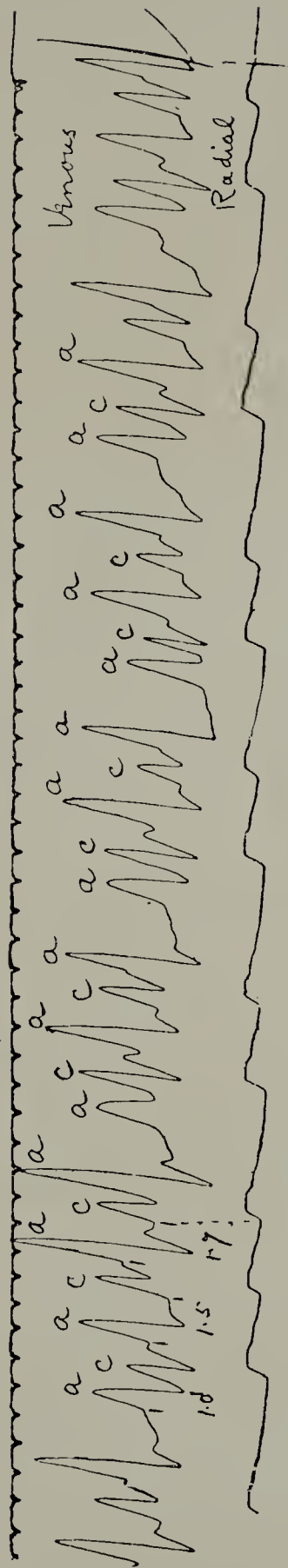


Fig. 68. Polygraphic curve from a case of exophthalmic goitre, recovering from an operation in which partial thyroidectomy was performed. Ventricular beats are dropped after each second or third complete cycle. Leading up to the failure of response, the *a-c* interval shows conspicuous widening. (Possibly vagal heart-block; the heart had been previously damaged by rheumatic fever, (full curves are given in *Quart. Journ. Med.*, 1910-11).

Evidences of contraction of the left, as opposed to the right auricle, are often obtained in the apex curves, and have been seen also in curves taken from the œsophagus,¹⁵ a method in which the recording tambour lies against the posterior aspect of the left auricle.

The most perfect of all methods of demonstration is the *electrocardiographic*, first used for this purpose by Einthoven.² By means of it the separate contractions of auricle and ventricle are recorded with ease and their time relationships to each other are readily shown (Fig. 71-76).

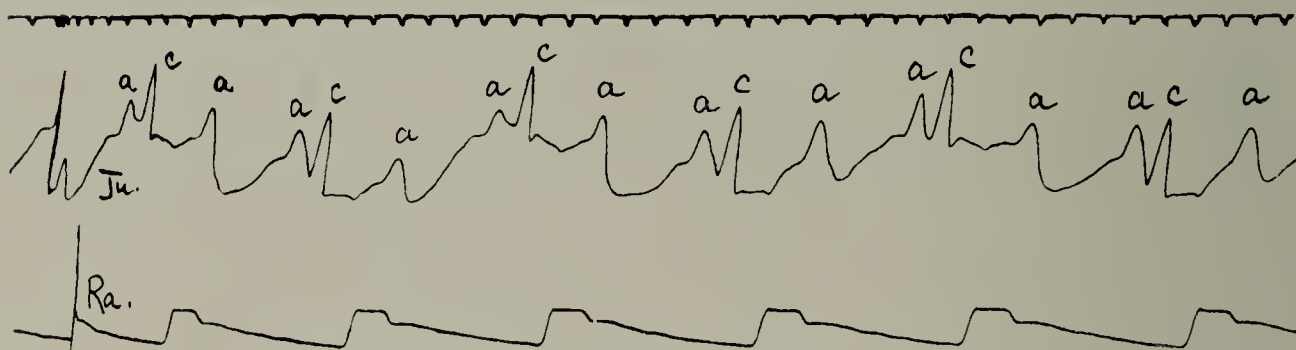


Fig. 69. Venous and radial curve from a patient presenting 2 : 1 heart-block.

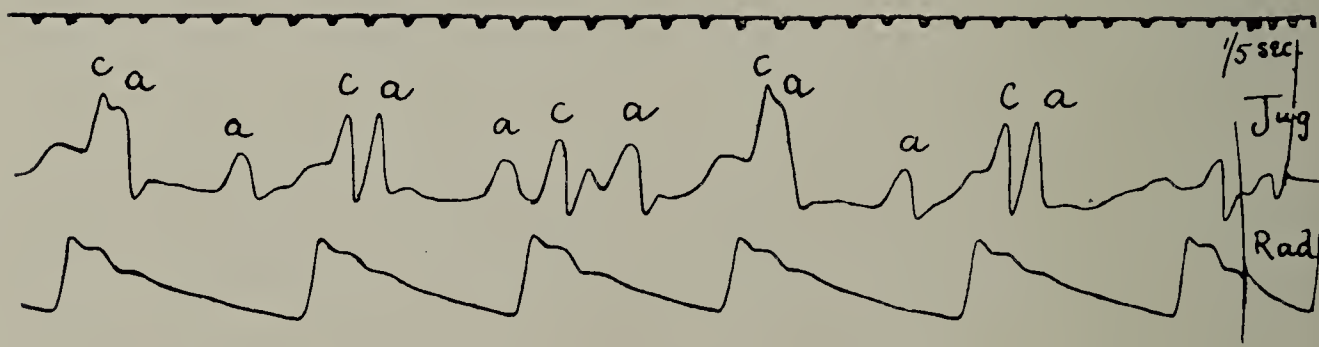


Fig. 70 (Windle, *Heart*, 1910-11, II, 104). Venous and radial curves in complete dissociation. The *a* waves fall in haphazard relationship to the ventricular systoles. The *a* waves themselves are placed in regular sequence, as are also the radial beats.

It has been shown beyond dispute that those grades of heart-block which are observed in the experimental investigations of the cold-blooded and mammalian heart also occur in man in pathological conditions.

The earliest evidence of impaired conductivity presents itself clinically, as in the lower animals, by an increase of the *As-Vs* interval (Fig. 66, 67, 68, 71 and 73), which, as we have seen, is indicated by the venous and electrocardiographic records (*a-c* and *P-R* intervals). In the pathological state the *a-c* interval may increase to at least .6 sec. (normal .1 to .2) and the *P-R* interval has been seen to increase to .5 sec. (normal .12 to .17 sec.).

Dropped beats (Fig. 68 and 72) are found at a more advanced stage and are usually foreshadowed by a temporary increase* of the conduction intervals (Fig. 68). Still later the auricular and ventricular rhythms may be found to stand in 2 : 1, 3 : 1, 4 : 1 ratios, etc. (Fig. 69 and 74). Finally the rhythms are found to be dissociated; the upper and lower chambers contract at entirely independent rates (Fig. 70, 75 and 76).

In partial heart-block simple ratios are most commonly encountered, yet a number of more complex relationships have been described, and particular attention has been paid to the factors influencing the transition of a heart-block of lower into one of higher grade or the reverse. While the observations are numerous, the evidence is still conflicting, and the subject, though an important one, is hardly ripe for full discussion.

In entering into detail we may confine ourselves to a single example of a transitional form of partial heart-block. By so doing our attention will be sufficiently directed for the time being to the more important factors influencing changes from one grade to another. Further references to the subject will be found in Chapters XIX and XXII.

The nature of alternating 1 : 1, 2 : 1 and 2 : 1, 3 : 1 ratios.

In certain cases of heart-block, periods are met with in which the mechanism wavers between a lower grade and a higher grade of heart-block. Thus it may be found that for short intervals 1 : 1 and 2 : 1 phases or 2 : 1 and 3 : 1 phases succeed each other in an alternate manner. An example of 2 : 1, 3 : 1 alternating ratios is shown in the first four cycles of Fig. 77. Disturbances of this kind are associated with notable variations in the *As-Vs* intervals. The interval following the shorter pause is relatively long; the interval succeeding the longer pause is relatively short. This increase or decrease of the *As-Vs* period, according to the length of the ventricular beat which precedes it, serves as an explanation⁶ of the curious alternation of rhythm in question. For instance, supposing (in Fig. 78) that the heart is beating in simple and continuous 2 : 1 sequence (cycles 1 and 2) and that one second separates each pair of the regular auricular beats, it will be obvious that as a result of the cycle in which response occurs (*a*) a hindrance to the passage of impulses from auricle to ventricle is engendered, and that the obstruction (indicated by the black triangle *R*) is maintained for at least one second. This is known, for there is no response to the second auricular cycle (*b*). On the other hand, the refractory phase (*R*) is of less duration than two seconds, for there is a response to the third auricular cycle (*a'*). The period of its termination lies between one and two seconds from its onset. Now suppose that the ratio is tending to break down from 2 : 1 to 3 : 1, then it should be clear that the point at which the hindrance to the passage of impulses in the 2 : 1 period vanishes (the end of the refractory phase) lies in the immediate neighbourhood of the onset of the third auricular cycle. That is to say, the refractory phase is somewhat less than two seconds. When the heart is in such a condition, that the duration of the refractory phase is almost equivalent to the distance which separates auricular contractions producing responses, the ratio of auricular and ventricular rhythms is in an evidently unstable condition, and a slight change either in conduction or in the auricular rate will produce a marked alteration of the mechanism.

Where there is also a tendency to variation in the length of *As-Vs* intervals, those have to be taken into account, and their examination proves them to be significant; for they in themselves increase or decrease the relative duration of the refractory phase. As a result of a slight increase of auricular rate (diagrammatised in the figure) the auricular contraction *c*² falls within the refractory phase (represented as constant). The additional pause, which the

* Instances of partial heart-block in which the *As-Vs* interval is constantly of normal length are not uncommon.^{4 & 10}

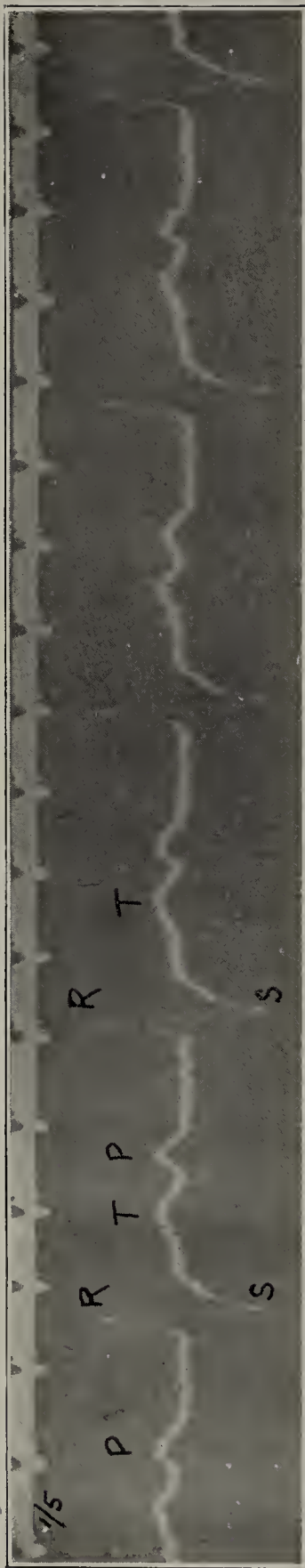


Fig. 71.

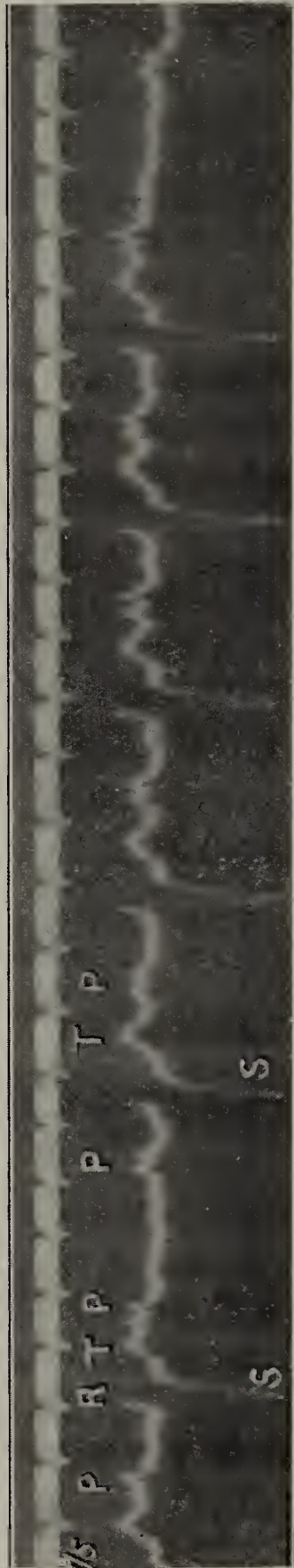


Fig. 72.

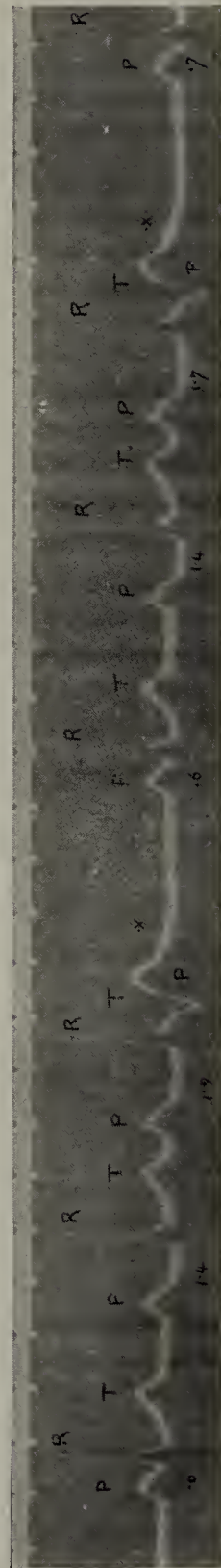


Fig. 73.



Fig. 74.

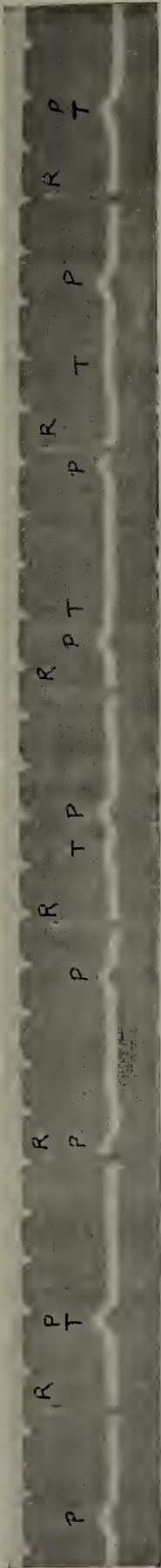


Fig. 75.

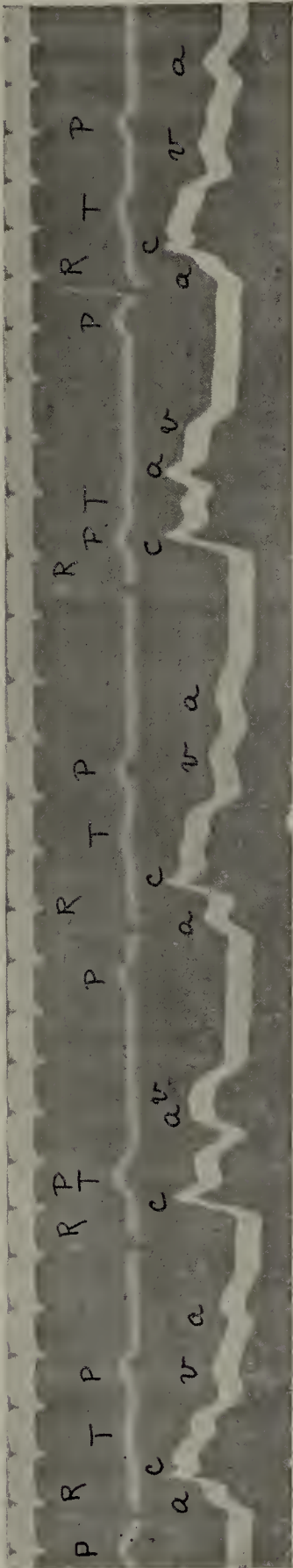


Fig. 76.

Fig. 71 ($\times \frac{5}{2}$). An electrocardiogram from a case of arterio-sclerosis exhibiting a partial grade of heart-block. The P-R interval exceeds .3 sec., as opposed to the normal interval of .12 to .17 sec..

Fig. 72 ($\times \frac{5}{2}$) (*Turnbull, Heart, 1909-10, II, 21*). An electrocardiogram from a case of mitral stenosis under the influence of strophanthus. The P-R interval is prolonged to nearly .3 sec., and a response is occasionally missed.

Fig. 73 ($\times \frac{5}{2}$) (*Heart, 1910-11, II, 127, Fig. 10*). An electrocardiogram from a case of mitral stenosis, showing a gradual increase of P-R interval from .08 to .34 sec.. Certain of the intervals are so short (*i.e.*, .12 sec.) that it is probable that some of the ventricular beats are "escaped" contractions.

Fig. 74 ($\times \frac{5}{2}$). Electrocardiogram showing the passage of a 1:1 to a 2:1 rhythm.

Fig. 75 ($\times \frac{5}{2}$) (*Windle, Heart, 1910-11, II, 105*). Complete dissociation. The ventricular rate, 63, is quite exceptional. Superimposition of P upon R and T is clearly seen in the figure.

Fig. 76 ($\times \frac{5}{2}$) (*Windle, Heart, 1910-11, II, 105*). Simultaneous electrocardiograms and venous curves from the same case. Showing the relationships of a and P waves, c and R, and v and T waves.

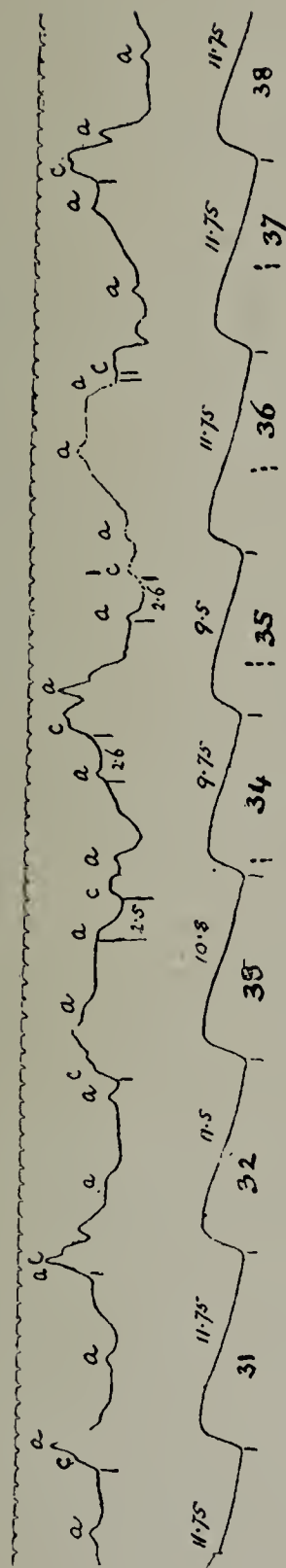


Fig. 79 ($\times \frac{3}{4}$) (*Journ. of Physiol.*, 1908, xxxvii, 449). Mixed response; polygraphic curve. The upstrokes of the first three radial cycles have no fixed relationship to auricular waves, the preceding pauses are of constant length; the beats are due to stimulus production in the ventricle. The upstrokes of the next three cycles (34, 35 and 36) are due to transmitted impulses from the auricles. The last two (37 and 38) belong to complete heart-block, as does also the pause of 36. Note the lengths of pauses 31, 32, 36, 37 and 38, and compare with Fig. 77 and 80, which are from the same case. The numbers below the cycles indicate the positions of the beats in a continuous tracing.

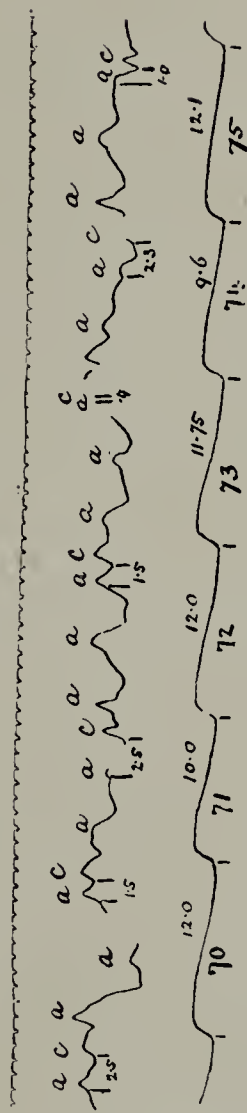


Fig. 80 ($\times \frac{5}{8}$) (*Journ. of Physiol.*, 1908, xxxvii, 450). Mixed response; polygraphic curve. The first three cycles belong to 2:1, 3:1 ratio. The upstroke of cycle 73 is due to a transmitted impulse from auricle. Cycle 73 is terminated by the occurrence of a spontaneous ventricular beat. Cycle 74 closes with a beat due to the auricle. Cycle 75 is a 3:1 cycle, and is the first of a series of cycles belonging to a period of 2:1, 3:1 ratio.

tissues enjoy as a consequence, leads to a reduction of the next *As-Vs* interval (a^3), the refractory phase commences earlier and terminates earlier and permits a response to a^4 . But the lengthened *As-Vs* interval which follows now delays the refractory phase and once more there is a failure of response to the third cycle (c^1). In this manner we may readily explain the production of a 2 : 1, 3 : 1 alternating ratio.

Examples of transitions from one grade of heart-block to another are given in Fig. 77, 79 and 80, which are accompanied by brief explanatory legends.

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- ¹¹ Evidence for the presence of *a* waves on the radial curves will be found in the following articles :—MACKENZIE, *Brit. med. Journ.*, 1905, I, 519; FRANÇOIS-FRANCK, *Archiv. d. Physiol.*, 1890, XII, 395; DRAPER, *Heart*, 1910-11, II, 95.
- ¹² References to auricular sounds of heart-block :—GALABIN, *Guy's Hosp. Rep.*, 1875; GIBSON, *Edin. med. Journ.*, 1905, N.S., XVIII, 9; FINNY, *Brit. med. Journ.*, 1906, I, 967; and many other writers.
- ¹³ The separate auricular movements are reported to have been seen radiographically by :—RITCHIE, *Proc. roy. Soc. Edin.*, 1905, XXV, 1085; FINNY, *Brit. med. Journ.*, 1906, I, 967; BROUARDEL and VILLARET, *Archiv. d. Méd. expér.*, 1906, XVIII, 252; and other writers.
- ¹⁴ A number of cases of heart-block, fully described with venous curves, will be found in the articles referred to in the succeeding chapters, and in the papers of :—RIHL, *Zeitschr. f. exper. Pathol. u. Therap.*, 1905, II, 83; ASCOLI, *ibid.*, 1907, IV, 185; VOLHARD, *Deutsch. Archiv f. klin. Med.*, 1909, LCVII, 348; a summary of previous reports and full bibliography is given by PLETNEW, *Ergeb. d. inn. Med.*, 1908, I, 47-67.
- ¹⁵ Full references to the œsophageal curves will be found in "Die funktionelle Herzdiagnostik." JANOWSKI, Berlin, 1910.

CHAPTER X

THE CAUSATION OF CLINICAL HEART-BLOCK.

The post-mortem findings.

THE present-day conclusion, that a very large percentage of all cases of clinical heart-block results from lesions of the auriculo-ventricular junctional tissues, is founded upon the experimental demonstration of the chief function of these tissues, namely, the conduction of impulses from auricle to ventricle. Pathological observations upon cases of clinical heart-block have been numerous, it is true, but they would be insufficient to establish fully the conclusion arrived at, were it not for our knowledge of the experimental facts. The question cannot be regarded purely from this point of view; we ask morbid anatomy to confirm the proposition that a lesion which transects the bundle will be associated with clinical heart-block, for it is accompanied by heart-block in the experimental animal. And we enquire as to the percentage of bundle lesions in cases which present heart-block clinically. Disease undertakes but few simple experiments, and the evidence of the association of heart-block and bundle affections in man is already and necessarily intricate.

Lesions of the bundle as they occur in the human heart are never confined to it, and often there is a more or less widespread damage of the whole organ. It must not be forgotten that from the special standpoint of pathology the more circumscribed the damage the more valuable it is as evidence. Localised lesions consequently command particular attention.

In giving an account of heart-block in man and the morbid changes accompanying it, it is our duty to enquire also as to the relationship between the grade of heart-block and the extent of the damage observed.

The material which we have at our present disposal consists of some fifty clinical cases, many of them imperfectly reported or observed; these have been arranged under convenient headings, and are presented to the reader in tabular form.

An examination of the records permits of certain general statements.

Up to the present time no case of heart-block has been reported in which a subsequent examination of the bundle and its connections has shown these structures to be absolutely intact histologically. And, on the other hand, no instance of complete destruction of the bundle has been demonstrated in a case in which the passage of impulses across the junction is known to have occurred directly before death. These are the chief conclusions at which we are able to arrive. But it is clear from an

Table of cases of heart-block examined post-mortem.

I. Cases of complete dissociation.

Author.	Reference.	Lesion.	Histological (Confirmation.	Tracings show Heart-block.	Degree of Heart-block.	Remarks.
Ashton, Norris and Laven- son.	Amer. Journ. med. Sci., 1907, cxxxiii, 28.	Gumma interrupting bundle completely.	+	+	C.H.B.	
Barr.	B.M.J., 1906, II, 1122.	Bundle more than half obliterated.	+	+	C.H.B.	Tracings suffered by reduction. Original curve demonstrative. (Personally examined.)
Bünniger and Mönckeberg.	Deutsch. med. Wochenschr., 1908, 2293; "Unters- uchungen über das Atrio- ventrikulärbündel im menschlichen Herzens." Mönckeberg, Jena, 1908, s. 232 and 294.	Total break in bundle as a result of calcification.	+	+	C.H.B.	Pulse 30-45. Auricles beating 2-2½ times as fast. Fits in second case. Electrocardio- graphic and venous curve taken; showed undoubted dissociation in both in- stances. (Personal commu- nication.)
		Total break in bundle as a result of sclerosis.	+	+	C.H.B.	
Fleming and Kennedy.	Heart, 1910, II, 77.	Lymphocytic deposits in node and bundle (diphtheritic).	+	+	C.H.B.	
Gibson and Ritchie.	Edin. med. Journ., 1909, I, 315 and 507; Lancet, 1909, I, 533; B.M.J., 1909, I, 404.	Node and first part of A-V bundle markedly fibrous and calcified.	+	+	C.H.B.	
Hay.	B.M.J., 1905, II, 1034.	Stretching of A-V bundle with partial obliteration.	—	+	C.H.B.	Pulse rate occasionally reached 80.
Hervheimer and Kohl.	Deutsch. Archiv f. klin. Med., 1910, xcvi, 330.	Infiltration of bundle and branches (left especially).	+	+	C.H.B.	

Hoffmann.	Deutsch. Archiv f. klin. Med., 1910, c, 172.	Chronic inflammatory lesion. Fibroid and calcareous changes in bundle.	+	+	C.H.B.
James.	Amer. Journ. med. Sci., 1908, CXXXVI, 471.	Deep ulcer invading bundle region in left side of ventricular septum.	+	+	C.H.B.
Krumbhaar.	Archiv. intern. med., 1910, v, 583-595.	"Occasional slight increase of connective tissue; no greater than is found in other hearts that have never exhibited any heart-block."	+	+	C.H.B.
Magnus-Alsleben.	Zeitschr. f. klin. Med., 1910, LIX, 82.	Parenchymatous degeneration of bundle (diphtheria).	+	+	C.H.B.
Pibram, Kahn and Koch.	Prag. med. Wochenschr., 1910, 233; Berl. klin. Wochenschr., 1910, 1108.	Slight fatty change in node and bundle, lymphocytic deposits in latter and in vent. septum.	+	+	C.H.B. Possibly incomplete at times.
Vollhard and Fahr.	Deutsch. Archiv f. klin. Med., 1909, xcvi, 348 (<i>Case XII</i>); Verhandl. d. Deutsch. pathol. Gesel., 1910, 105.	Fibrosis of bundle and its branches, right especially.	+	+	C.H.B. Curves unpublished, stated to show unquestionable dissociation.
Vollhard and Mönckeberg.	Deutsch. Archiv f. klin. Med., 1909, xcvi, 348 (<i>Case VI</i>); "Untersuchungen über das Atrio-ventrikulärbündel im menschlichen Herzens." Mönckeberg, Jena, 1908, v, 300.	Fibrosis of junctional tissues, chiefly affecting the left division of bundle.	+	+	C.H.B.

II. Cases in which partial heart-block was alone observed.

Author.	Reference.	Lesion.	Histological Confirmation.	Tracings show Heart-block.	Degree of Heart-block.	Remarks.
Cohn, Holmes and Lewis.	Heart, 1910, II.	Fibrosis and vascularisation destroying large proportion of bundle.	+	+	Partial	Transient H. B. only.
Gerhardt.	Deutsch. Archiv f. klin. Med., 1908, xciii, 485.	Acute inflammation of bundle (rheumatic).	+	+	Partial	
G. A. Gibson.	B.M.J., 1906, II, 1113.	Sclerosis and separation of muscle fibres.	+	+	Partial	
Heineke, Müller and Hösslin.	Deutsch. Archiv f. klin. Med., 1908, xciii, 460.	Gummatous infiltration of bundle.	+	+	Partial; possibly complete later.	

III. Cases in which partial and complete heart-block were present.

Griffith and Cohn.	Quart. Journ. Med., III, 126.	Syphilitic lesion partially obliterating the bundle.	+	+	Partial and C.H.B.	
Hay and Moore.	Lancet, 1906, II, 1271.	Right coronary narrowed. Central body atheromatous. Partial obliteration of bundle.	+	+	Partial and C.H.B.	

IV. Case in which with complete bundle destruction partial heart-block was present some weeks before death.

Heineke, Müller and Hösslin.	Deutsch. Archiv f. klin. Med., 1908, xciii, 460.	Complete destruction of bundle by sclerosis, calcification and hæmorrhage.	+	+	Partial	Curve obtained six weeks before death.
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V. Cases in which the grade of heart-block was not clear.

Bramwell.	B.M.J., 1909, I, 995.		Bundle well-nigh obliterated by calcareous nodule and fibrosis.	+	+	Probably C.H.B.	Tracing imperfect.
A. G. Gibson.	Quart. Journ. Med., I, 182.		Partial obliteration of bundle by fibrosis and fat deposit.	+	+	?	Pulse 35. Fits. Curve shows heart-block, but grade uncertain. (Personal examination.)
Jellinek and Cooper.	B.M.J., 1908, I, 796.		Anæmic necrosis of muscle in neighbourhood of bundle. ? Gonorrhœal.	+	+	Probably C.H.B.	
Nagayo.	Zeitschr. f. klin. Med., 1909, LXXVII, 495.	Med.,	Hard tumour in septum, fibrous and calcified. Bundle pressed on, flattened and atrophic.	+	--	?	Curves are said to have shown C.H.B.
Stengel.	Amer. Journ. med. Sci., 1905, CXXX, 1083.	Sci.,	Endocardial lesion in upper part of left ventricle; also in septum. Said to involve bundle.	.	+	Probably partial.	Tracings defective. Pulse rate 16-147.
Turrell and Gibson.	B.M.J., 1908, II, 1486.		The bundle represented by a few isolated strands.	+	+	?	Tracing imperfect. Slow pulse and fits.
Vaquez and Esnein.	Bull. et mém. d. l. Soc. Méd., 1907, XXIV, 78.	l. Soc. Méd.,	Gummatous sclerosis affecting the bundle.	+	+	? C.H.B.	Tracing imperfect. Pulse 60-36. Syncopal attacks.

VI. Cases of Adams-Stokes' syndrome in which the presence of heart-block has not been fully established.

Asehoff.	B.M.J., 1906, II, 1103.		Severe sclerosis of arteries traversing node and principal part of bundle.	+	—	?	Adams-Stokes' syndrome stated to have been present. (Cases of Hering's.)
Asehoff.	B.M.J., 1906, II, 1103.		Marked fatty deposits in node and bundle.	+	—	?	
Beck and Stokes.	Archiv. intern. Med., 1908, II, 277.	Med.,	Marked calcareous infiltration of the A-V groove. Pressure upon and atrophy of bundle.	+	+	?	Tracing defective. Pulse 26-32. Fits accompanied by cessation of ventricular beat.

VI. Cases of Adams-Stokes' syndrome, in which the presence of heart-block has not been fully established (*continued*).

Author.	Reference.	Lesion.	Histological (confirmation).	Tracings show Heart-block.	Degree of Heart-block.	Remarks.
Beeson.	Journ. Amer. med. Assoc., 1908, I, 188.	Calcareous tumour in ventri- cular septum in neighbour- hood of bundle.	—	—	?	Slow pulse. Fits, rapid venous pulsation. (Insufficient evi- dence.)
Bishop.	Amer. Journ. med. Sci., 1910, CXXXIX, 62.	Calcareous nodule situated pre- cisely in the path of the bundle.	—	—	?	Tracing defective. Pulse slow (18-40). Vertiginous and syn- copal attacks. Rapid venous pulse.
Butler.	Amer. Journ. med. Sci., 1907, CXXXIII, 715.	Bundle showed fatty infiltration and degeneration. Atrophy to a fifth normal size.	+	—	?	Pulse 6-42; venous pulse rapid. Epileptiform seizures.
Chapman, Keith and Miller.	Lancet, 1906, II, 219; and 1906, II, 1429.	Upper half of bundle completely destroyed (gummatous).	+	—	?	Pulse 32-54. Syncopal attacks.
Fahr.	München. med. Wochen- schr., 1907, LIV, 636; Virchow's Archiv, 1907, CLXXXVIII, 562.	Sclerosis at division of bundle. Left branch gone; right par- tially destroyed.	+	—	?	Adams-Stokes' syndrome stated to have been present.
Handford.	B.M.J., 1904, II, 1745.	Gummata invading septum and ring.	—	—	Probably partial.	Pulse 80-90. Long pauses of 5- 15 sec.. Fits.
Handwerck.	München. med. Wochen- schr., 1909, LVI, 916.	Gumma in intra-auricular septum, invading bundle region.	—	—	?	Slow pulse (36-42), and syncopal fits. Venous pulse rapid.
Jagic.	Zeitschr. f. klin. Med., 1908, LXVI, 183.	Numerous gummata in ventri- cular septum, bundle region infiltrated.	—	—	?	Pulse 48-60. Fits of unconscious- ness. Rapid venous pulsations.
Karcher and Schaffner.	Berl. klin. Wochenschr., 1908, XLV, 1266.	Fibrosis and thinning of bundle (half destroyed).	+	—	?	Pulse 33-58. Fits. Rapid venous pulsations.

Keith and Flack.	Lancet, 1906, II, 359.	Gumma of septum of ventricle. A-V fibres involved.	+	—	?	Slow pulse. Fits of unconscious- ness with stoppage of heart. (Personal communication from Dr. Grünbaum.)
Löwenstein.	Verhandl. d. deutsch. pathol. Gesellsch., 1908, 160 (discussion).	Membranous septum shows a calcareous node at its lower border.	—	—	?	Pulse 27.
Luce and Fahr.	Deutsch. Archiv f. klin. Med., 1902, LXXIV, 370; München. med. Wochen- schr., 1907, LIV, 636; Virchow's Archiv, 1907, CLXXXVIII, 562.	Tumour penetrating ventricular septum and completely de- stroying bundle (gumma).	+	—	?	Pulse 28-48. Fits, accompanied by pulse slowing.
Mönckeberg.	"Untersuchungen über das Atrioventrikulärbündel im menschlichen Her- zens," Jena, 1908, 228.	Sclerosis and calcification in neighbourhood of bundle. No absolute loss of continuity.	+	—	—	Slow pulse and fits.
Mosbacher.	München. med. Wochen- schr., 1908, LV, 1983.	Fibrous infiltration of septum in bundle region.	—	—	?	Slow pulse. Fits, with cessation of pulse beats.
Rendu.	Bull. et mém. d. l. Soc. méd., 1895, XII, 381.	Gumma of ventricular septum, high up.	—	—	?	Pulse 30-40. Giddy attacks. Vein pulsating rapidly.
Robinson.	Bull. Ayer clin. Lab. Phila- delphia, 1907, No. 4, 1.	Gumma invading ventricular septum and bundle region.	—	—	?	Pulse 30-40. Sudden death.
Schmoll.	Deutsch. Archiv f. klin. Med., 1906, LXXXVII, 554.	Sclerosis causing complete atrophy of bundle.	+	—	?	Pulse 28-40. Fits, accompanied by cessation of ventricular beats. Venous pulsation rapid.
Sendler.	Centralbl. f. klin. Med., 1892, XIII, 642.	Hard grating tumour (fibroma) in upper part of ventricular septum.	—	—	?	Pulse 22-40. Fits of unconscious- ness.
Vickery.	Boston med. and surg. Journ., 1907, CLVII, 823, and 1908, CLIX, 435.	Diffuse fibrous and calcareous change in ventricular septum, involving bundle region.	+	+	?	Tracing defective. Pulse 30-35. Convulsions.

examination of the records that, excepting the cases in which the bundle is completely divided, it is impossible to estimate, from histological examination, the antecedent degree of functional impairment. There are cases on record in which gross lesions have been described, in which the bundle has undergone considerable structural alteration, in which the conducting path is reduced to a fraction of its original diameter, or in which one main division has been completely, and the other partially divided, but where only relatively slight grades of heart-block or temporary defects of conduction have been observed. There are cases in which, with far slighter degrees of apparent damage, dissociation has been complete and persistent. Nay, further, there are cases of complete dissociation in which the bundle lesions have been less conspicuous than in hearts in which no conduction disturbances could be demonstrated (Krumbhaar). The meaning of the first group is readily appreciated, for we are aware that a large number of the bundle fibres, or of the fibres of the main divisions, may be obliterated, and no change in sequence ensue. The second group is of special interest, for structural changes in the bundle are by no means infrequent in hearts in which the sequence of contraction is normal. In the hearts of elderly subjects such changes are the rule,⁹ and they were found in 70 per cent. of all cases of heart affection examined by Sternberg¹³ (in a total examination of 72 hearts). The affection of the tissues under these conditions may be of as high an apparent grade as in instances in which complete dissociation has been present. The apparent lack of harmony results from our inability to recognise the functional capacity of the undamaged fibres by the closest possible inspection of them. It arises from our lack of appreciation of the factors which may influence conduction through unbroken strands.

Nevertheless it may be stated that, where a lesion productive of heart-block is found, it usually constitutes the most conspicuous injury in the whole organ. More or less circumscribed areas of disease are not uncommon; they are of varied nature. The most frequent is the gumma, of which one or more may be present in the septum, or a somewhat less clearly defined infiltration or scarring of syphilitic origin. Local foci of fibrosis, combined or not with calcareous degeneration, are not infrequent in the neighbourhood of the membranous septum or in the lowest level of the auricular septum. A calcareous nodule in the path of the bundle; ulceration eating into it; an atheromatous degeneration in the neighbourhood of the central fibrous body, or an area of necrosis at the upper part of the ventricular septum, and involving the tissues in question (the last two lesions probably of arterial origin) have been described.

A number of the remaining descriptions are of a far less definite nature; an increase of the connective tissue of the bundle or node, atrophy of the bundle as a result of pressure or stretching are examples of this kind.

Other instances may be cited in which, as part of general myocardial change, the junctional tissues are affected by lymphocytic deposits, fatty degeneration or widespread fibrosis. In regard to the more diffuse processes, it has been stated that fatty and fibroid changes may show a peculiar predilection for the specialised tissues. Where there is no reason to suspect antecedent heart-block,⁵ the whole heart is usually more uniformly affected.¹³

The relationship of heart-block to rheumatic infection of the heart and mitral stenosis.

The histological examinations of the bundle have been confined for the greater part to cases of persistent heart-block of high grade, and instances of slow pulse associated with syncopal or epileptic seizures. These patients form a distinct clinical group, which is comparatively small.

Heart-block of slight grades, and temporary or permanent in its course, is more common, and is generally a sequel of rheumatic infection of the heart; it is especially associated with mitral stenosis. The distinction between the two groups spoken of is an important one.

It is true that heart-block of the higher grades, and accompanied by the graver disturbances, has been reported during the course of rheumatic fever; but they are cases which are, generally speaking, of acute onset (as in the cases of Gerhardt and James, which are included in the tables), and in a measure accidental. The group upon which emphasis is laid at the present time is one originally described by Mackenzie.⁶ A very large percentage of cases of rheumatic heart disease present evidences of impaired conduction, manifested by a widening of the *a-c* and *P-R* intervals. This is especially the case in patients the subject of advanced mitral stenosis. The evidence of deficient conduction in those cases of mitral stenosis which present an auriculo-ventricular sequence of chamber contraction is probably as high as 15 per cent. in out-patients. In in-patients it is certainly higher.

The mechanism of the heart in 106 cases of mitral stenosis, collected in an out-patient department.

	Total.	<i>a-c</i> less than ·2 sec.	<i>a-c</i> ·2 sec.	<i>a-c</i> greater than ·2 sec.
Auriculo-ventricular sequence ...	84	33	37	14
Auricular fibrillation ...	22			

The structural changes underlying the functional defect are imperfectly known. A single examination has been reported by Mönckeberg. The details of this case are given over page.

We have considerable information as to the histological appearances of the junctional tissues in cases of advanced mitral disease;^{4, 5, 8 & 12} the

<i>Author.</i>	<i>Reference.</i>	<i>Lesion.</i>	<i>Histological confirmation.</i>	<i>Tracings said to show Heart- block.</i>	<i>Degree of Heart-block.</i>	<i>Remarks.</i>
Mönckeberg.	Ergebn. d. allg. Patholl. u. patholog. Anat., 1910, XIV, 687.	Marked lymphocytic infiltration of the node (mitral sten- osis).	+	+	? partial.	Occasional heart- block observed and registered.

accounts are in general agreement. The heart is the subject of chronic inflammatory processes, lymphocytic infiltration, increase of connective tissue, and diffuse fibrosis: and these changes often seem to fall with particular severity upon the specialised tissues, the sino-auricular and auriculo-ventricular node and bundle. An isolation of the group is the more imperative, because the mechanism of the heart so affected shows certain definite reactions to cardiac poisons, which we shall proceed to consider.

The action of drugs of the digitalis group upon heart-block.

We are aware that poisonous doses of digitalis administered intravenously to healthy animals may result in heart-block.^{2 & 14} A similar action in man has not been demonstrated, for similar dosage cannot be employed, but we know that full clinical doses have no such effect upon the healthy heart. The heart which has been affected by rheumatism and which manifests signs of impaired conduction presents a peculiar idiosyncrasy to the drug, as Mackenzie has clearly shown. *Where the "a-c" interval is already increased* the administration of digitalis, strophanthus or squills causes a further prolongation of the interval, and the condition may progress to one of more decided heart-block, dropped beats, 2:1 rhythm, or on rare occasions even dissociation.¹⁵ It would appear that in certain instances, at all events, this action of digitalis is an indirect one and through the vagus, for the administration of atropine may reduce the heart-block to its original grade.¹⁵ That digitalis has a profound action upon the vagus has been established both experimentally and clinically, and, according to Tabora,¹⁴ digitalis increases the susceptibility of the experimental heart towards a manifestation of heart-block when the vagus is excited.

The vagus as a cause of clinical heart-block.

It has been said that vagal stimulation in animals may result in heart-block (Chapter VIII; and Fig. 81). But the heart-block so produced is usually of a very temporary character. Can inhibitory influences be

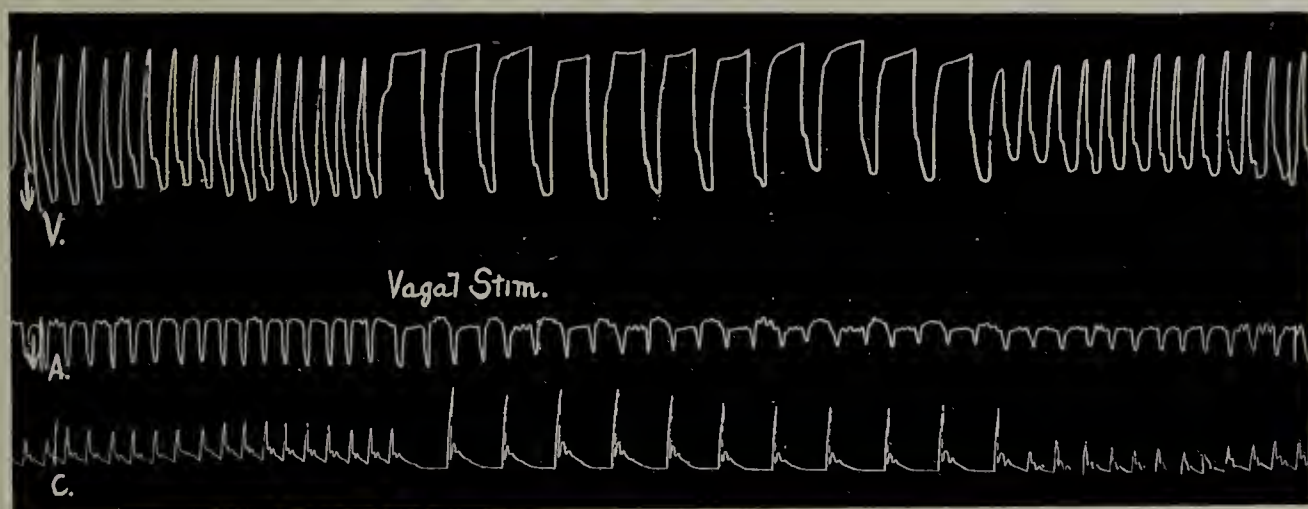


Fig. 81 ($\times \frac{1}{2}$). Myocardiographic curves (V =ventricle, A =auricle) and Hürthle carotid pressure curve (C) from a dog, showing an effect of vagal stimulation. It should be noted that the myocardiographic levers are writing downwards. At the onset of vagal stimulation, 2:1 heart-block is established and continues during the stimulation. At the same time there is slight auricular slowing.

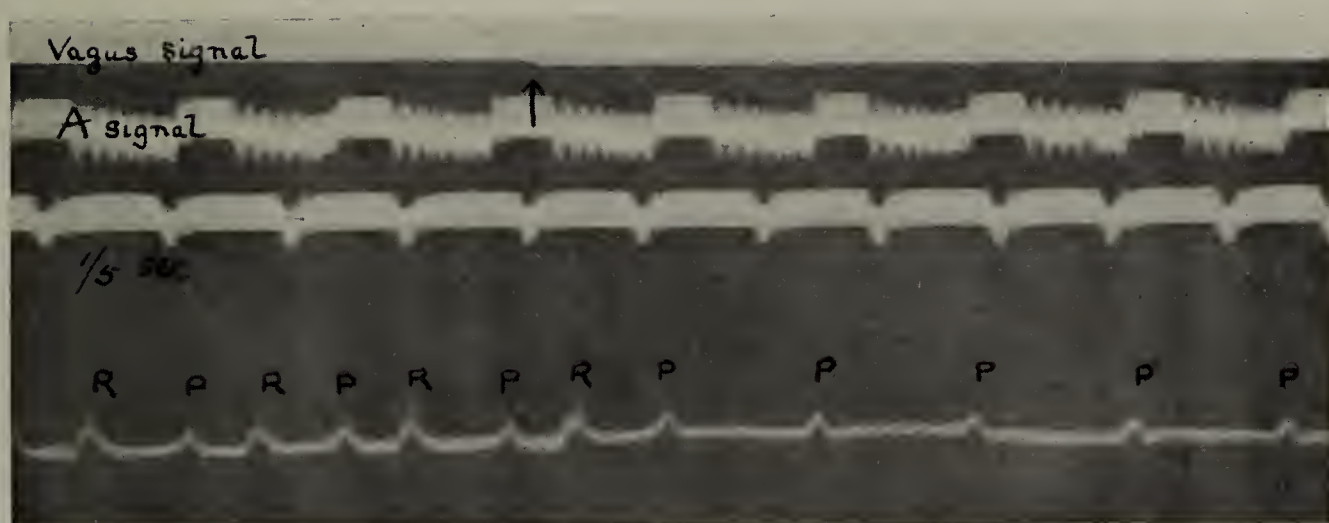


Fig. 82 (*Quart. Journ. Med.*, 1910-11, iv, 145, *Fig. 8*). An electrocardiogram from a cat during asphyxial heart-block. When a stage at which the P - R interval was prolonged had been reached, the vagus was stimulated (at the arrow and upper signal). From the point of stimulation to its termination there were no responses of ventricle to auricle. The auricular rate was maintained at a constant rate by interrupted induction shocks applied to it (A signal).

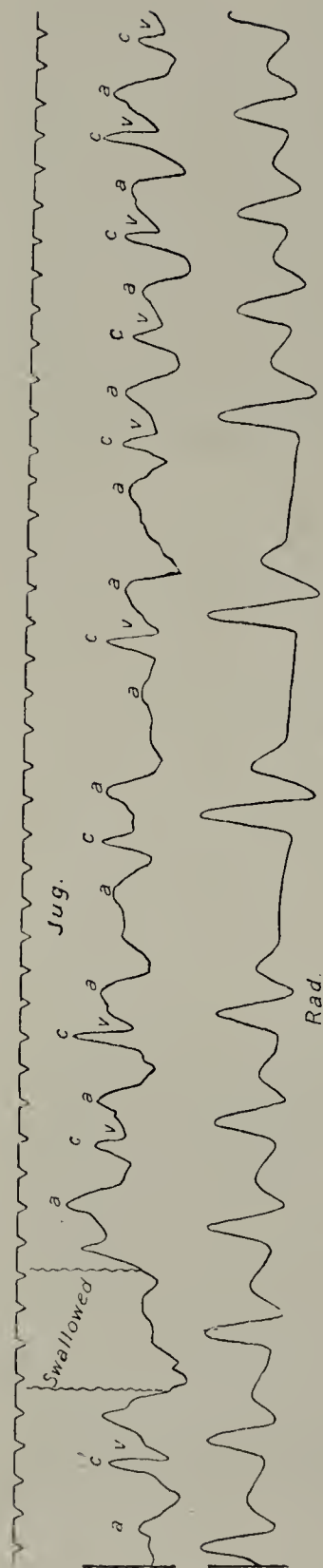


Fig. 83 (Mackenzie, *Brit. Med. Journ.*, 1906, II, 1110.) A polygraphic tracing from a case of partial heart-block (prolonged *a-c* interval), showing the effect of swallowing upon the heart's mechanism. After an interval of three heart cycles, temporary 2:1 heart-block is produced. A clinical example of an increase in the degree of heart-block as a result of vagal inhibition.

instanced to account for grave and prolonged disturbances of conduction in man? The answer to this question is probably in the negative. It is certainly in the negative where the majority of cases of chronic heart-block are concerned. For as we have seen there is sufficient demonstrable change in the junctional tissues either fully to account for the observed disturbances of mechanism, or to render this region more especially suspect. Atropine, a drug which paralyses the vagal nerve endings, has been repeatedly administered in full doses in clinical heart-block. Generally speaking it even fails to decrease the grade of disturbance. It has never been known to abolish the whole hindrance to conduction. There are, however, one or two instances in which the relief of heart-block by atropine has been observed. In Volhard's case,¹⁵ atropine abolished an increase of heart-block, produced by digitalis; so also¹⁰ in Rihl's case.*

No clear clinical instance of the initiation of heart-block by inhibitory influences has been published; but there is, as we have seen, definite evidence that such influences may increase the grade of a pre-existing defect of conduction.

The influence of the vagus upon tissue bridges in the heart, through which impulses are transmitted with difficulty, was first noted by Gaskell in his experiments upon the cold-blooded animals. The increase of heart-block as a result of vagal stimulation has also been observed in experimental mammalian heart-block, induced by means of the clamp, or by means of asphyxia (Fig. 82).

* There is a single observation by Barringer¹ upon a case of temporary dissociation, where an injection of atropine was followed by quickening of the ventricle (but the curve taken after the atropine injection does not demonstrate complete restoration of conduction).

A more striking clinical example of this action of the vagus could not be found than that published by Mackenzie.⁷ He observed that swallowing, which as is well known provokes an inhibitory cardiac reflex under normal conditions, produced failure of ventricular responses in a patient, who previously exhibited prolongation of the *a-c* interval. A curve from this patient is reproduced. Pressure upon the vagus in the neck has been noted by several observers to have a like result under similar circumstances.^{10 & 15}

The contents of this chapter may be summed up in the statements that auriculo-ventricular heart-block as it has been observed clinically has been due in the great majority of cases, if not all, to structural alteration in the auriculo-ventricular bundle, and that digitalis and vagal heart-block have been usually, if not always, the outcome of the action of the drug or the nerve upon tissues already deficient in the power of conduction.

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CHAPTER XI.

THE IDIO-VENTRICULAR RHYTHM: HOMOGENETIC AND HETEROGENETIC HEART BEATS.

The idio-ventricular rhythm.

IN preceding chapters the main facts in relation to experimental and clinical heart-block are set forth in detail, and it has been seen that when the grade of heart-block is severe the ventricle exhibits its own spontaneous rhythm.

The rhythm acquired by the ventricle under these circumstances, the so-called *idio-ventricular rhythm*, has received very careful study, but our knowledge of it is still in the stage of development. It is customarily regarded as dormant under natural circumstances, for it shows no signs of activity. But the degree of its dormancy is still *sub judice*. If the connections between auricle and ventricle are severed abruptly, the mammalian ventricle usually remains quiescent for a long period; it subsequently develops a rhythm of its own which, at first inordinately slow, gradually accelerates. But, even when fully developed, the rate never approaches that of the rhythm generated in the auricle. It would seem from the work of Erlanger² and his associates that this "rhythm of development" as Gaskell³ calls it, is awakened at a rate which is largely controlled by the ventricle's state of preparedness for the call which is made upon it to fulfil its subsidiary function. The more rapidly the connection with auricle is broken the longer does the ventricle stand quiescent. And his observations suggest that under ordinary conditions the ventricular pace-maker is inactive, but that certain interferences partially or fully awaken it, so that it is ready to act the part of substitute when the impulses from the higher levels are eventually withdrawn.

When the bundle is broken by disease, the distal end of it undergoes no visible change, suggesting that it continues to functionate. The electrocardiographic records of dissociation have demonstrated that the ventricular beat has its origin in this structure, for the ventricular complex (Fig. 61 *I V* and 75) is of the type recognised as derived from a supraventricular impulse; that is to say, the contractions of the ventricle in this condition are of a nature compatible with the receipt of impulses along the usual channels (right and left divisions of the bundle) and with their receipt along these channels alone; the conclusion is tantamount

to a statement that the impulse is generated in the upper reaches of the distal segment of the divided tissues.

The idio-ventricular rhythm is said to show no sign of activity under ordinary circumstances for the reason that the muscle, in which the impulses are elaborated, is in receipt of extraneous impulses, which succeed each other more rapidly than do those which are intrinsic. Whether this is strictly true for the normal heart is a question as yet undecided but it is perfectly true that the sinus pace-maker and a ventricular area frequently build up impulses simultaneously, and that the rates of the independent rhythms, while showing great constancy individually, are widely separated. The recognised sinus rate in man is 72, while the usual ventricular rate is 30. If sinus and ventricle are generating distinct impulses at these rates in an undamaged heart, there is no difficulty in understanding the absence of response to the lower rhythm producing focus. For it can be shown experimentally that a contraction of the ventricle destroys a generating impulse in its own substance. A single induction shock applied to a ventricle responding regularly to intrinsic impulses, awakens a contraction of the ventricle; and at whatever point of diastole this new or interrupting beat falls, it is succeeded by a similar event, namely, a pause which is equal to the interval between two beats of the spontaneous rhythm,⁵ and the succeeding beat is in every way identical with a beat of the preceding rhythm (Fig. 84 and 85). The beat which follows the interruption has all the characteristics of an idio-ventricular beat and may be assumed to be of a similar nature. Thus while the ventricle is shown to be excitable during the whole of the diastolic intervals of its cycles, the impulse which gives rise to the spontaneous contraction requires a second and a half to prepare itself (assuming the rate to be 30 per minute and the length of systole to be .5 sec.), and this is so whether the spontaneous beat succeeds a contraction of a similar nature, or whether it follows an extrinsic impulse. But the extrinsic beat definitely disturbs the idio-ventricular impulse preparation, delaying it by precisely that interval of time which lies between the extrinsic beat and the systole which precedes it (*b* in Fig. 84). In other words, that portion of the time of preparation which occurs before the appearance of the interrupting beat is entirely lost. The phenomenon is regarded as the result of the destruction of the chemical products, upon which the expected spontaneous beat depends, and which are considered to be in process of formation. The interrupting beat obliterates the stimulus material which precedes it. We are brought to the conclusion that each spontaneous beat results from a gradual building up of stimulus material, a process which usually occupies at least a second and a half.

Now although, while the sinus rhythm fully dominates the ventricular, there is no clear evidence of impulse formation in the ventricle itself, yet when partial grades of heart-block are present, the evidences of a spontaneous ventricular rhythm appear from time to time, and in some instances

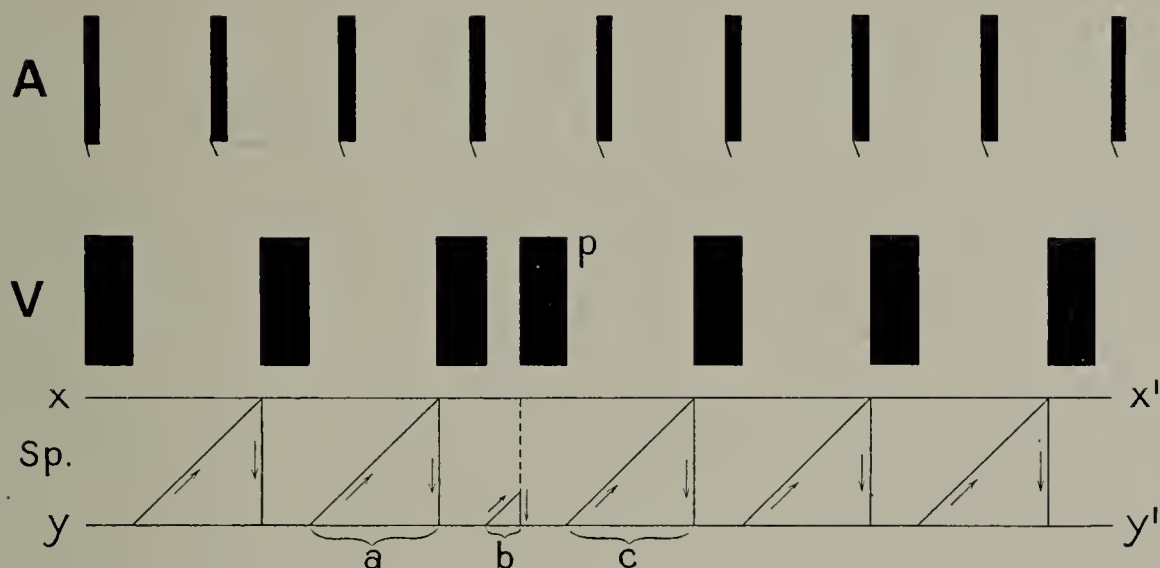


Fig. 84. A diagram illustrating the events in auricle and ventricle when complete dissociation is present. The vertical rectangles A and V represent contractions of auricle and ventricle respectively. The oblique lines placed below (Sp) represent stimulus production in the ventricle; the impulses are represented as built up at a uniform rate, and as discharged with each ventricular systole to which they give rise. The point of discharge lies at the line x, x' . The premature beat p destroys the stimulus material which has accumulated over the short interval b . The next systole follows a pause (c) which is equal to the interval following beats of the regular spontaneous rhythm (a).

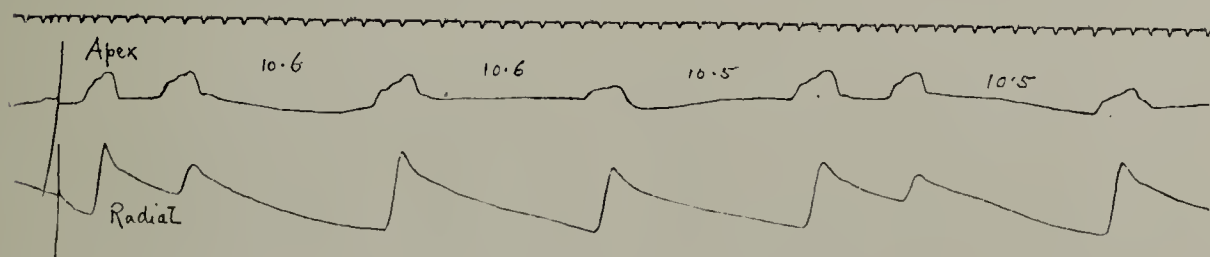


Fig. 85 ($\times \frac{2}{3}$). An apical and radial curve from a case of complete heart-block, showing two premature ventricular contractions. The pauses which succeed them are of the usual length; they are equivalent to those which succeed the usual rhythmic beats.

the conditions are remarkable. Heart-block of the 2 : 1 grade is common, but 3 : 1 heart-block is comparatively rare, and this is in part accounted for by the fact that a failure in the passage of two impulses in three so retards the ventricular rate that spontaneous impulses have time to develop in the ventricle and, giving rise to successive contractions, produce a temporary picture of complete heart-block ; a condition termed by Erlanger "relative complete heart-block," and illustrated in Fig. 78-80.

A notable example of ventricular "escape" has been recorded by Wenckebach,¹¹ and may be taken as a type. The mechanism of the heart was such that whenever, as a result of slowing, a sufficient time elapsed a spontaneous ventricular beat manifested itself and interrupted the otherwise sequential contraction of the heart chambers. The relative or absolute constancy of the preceding pause in an individual case is a characteristic of such beats.

The heart's mechanism in instances of the appearance of isolated idio-ventricular beats may best be appreciated by diagrammatising it. The auricular and ventricular contractions are indicated by the two upper rows of vertical rectangles (*A* and *V*) of the accompanying figure. Stimulus production is shown in the ventricle by the triangles at the bottom (*Sp.*). Throughout the diagram stimulus matter is represented as being built up at a constant rate, and as being destroyed at each ventricular systole. At only one point does a sufficient time elapse to allow of its full development ; at this point (*) in the diagram) it generates a spontaneous ventricular beat. Actual examples of escape are shown in Fig. 87, 88 and 89.

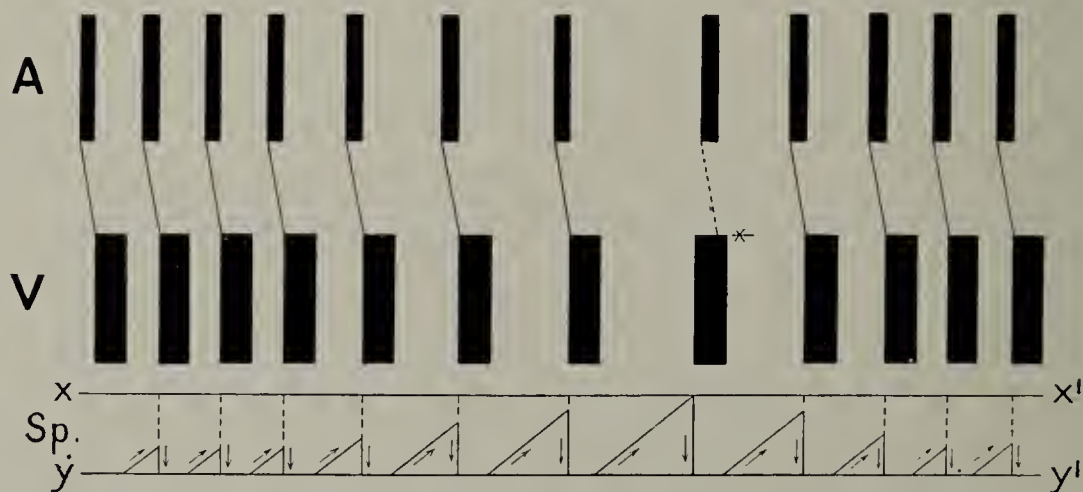


Fig. 86. A diagram illustrating ventricular escape, when the sinus rhythm is sufficiently retarded and when the conditions predispose to such escape. It is supposed that stimulus material (*Sp.*) is built up at a constant rate in the ventricle, but that as a rule the pauses are of insufficient duration to allow an accumulation to the critical point (the line *x*, *x'*) at which the growing impulses are discharged. At one point in the figure a spontaneous ventricular beat (marked with an asterisk) is represented, where the pause is longest.

Homogenetic Heart Contractions.

A full acquaintanceship with the idio-ventricular rhythm is of considerable importance, for it depends upon a type of impulse formation, which may be termed physiological or *homogenetic*. It is possible, if not probable, that many portions of the cardiac musculature are capable of giving rise to rhythms of this nature; such rhythms have been studied in isolated portions of muscle, for example, in the apex of the frog's heart and in strips of the terrapin heart. They are rhythms which arise spontaneously in areas of the muscle, which are in receipt of no extraneous impulses, and which are placed amid suitable and, so far as possible, natural nutritive surroundings.

They appear to be particularly associated with the so called primitive cardiac "rests." The observations of Gaskell³ have paved the way to an acknowledgment that such tissues are specially endowed with rhythmic functions. The normal rhythm of the mammalian heart, originating in the specialised tissue at the cavo-auricular junction is the first representative of the type under consideration. There are a number of observations, clinical and experimental, which suggest that the junctional tissues, more especially the auriculo-ventricular node, are occasionally the seat of rhythmic discharges. The idio-ventricular rhythm, arising in the distal end of a divided bundle is another example.

Contractions of the heart which may be spoken of as homogenetic present certain striking characteristics. They belong essentially to, and are but single integers in, a rhythmic series of similar contractions. The impulses upon which they depend are notable for the relative constancy of their time of preparation, and the time of impulse preparation is comparatively long.

But the heart muscle can also respond with an abrupt contraction to an external cause of irritation, and then, when the external irritant ceases to act, it resumes its slow homogenetic rhythm, after a appropriate pause. The abnormal beat with its peculiarly quick time-relation can be evoked experimentally by a known external cause, it has its precise counterpart in the premature beat which arises pathologically. It may even be repeated rhythmically. But whether there is a single beat or a regular beat in series the time-relations of the contractions always differ essentially from those of homogenetic contractions. Unlike the latter, they are confined to pathological conditions, and are classed in consequence as *heterogenetic*.

Heterogenetic contractions and their contrast with homogenetic beats.

A single induction shock of sufficient intensity applied to the auricular or ventricular surface, and in any part of the diastole of one or other chamber, awakens an active response. The period of latency between the application of the stimulus and the initial phases of the contraction amounts in the mammalian heart to a very small fraction of a second (frequently as small as .01 sec., and probably never greater than .03 sec.). The actual relationship of the stimulus to the functions of the heart muscle

is not known. But it seems apparent that either impulse formation is dispensed with, or that a phenomenally rapid impulse is constructed in the musculature. The period of rest in the excited ventricle is reduced to a minimum. A very similar difference may be traced between the physiological impulse and the premature contraction, as it is encountered in pathological conditions. Beats may be initiated spontaneously and prematurely in the ventricle at intervals of $\cdot 03$ sec., or less, from the cessation of the preceding contraction. Compare this interval with the preparation time of the idio-ventricular beat (usually at least 1.5 sec.) and the difference is remarkable. Yet both beats arise spontaneously in the ventricle. And there is a further and striking contrast between the two types of contraction. If the idio-ventricular impulse formation is active, the ventricle responds to it whenever a sufficient time elapses for the full development of an individual stimulus. In the instance of the premature and pathological type of beat, such a phenomenon is not encountered. When a premature beat occurs, it is customary to find but a short interval between it and the preceding systole, and it is followed by a pause which usually exceeds the distance between two beats of the sinus rhythm. Sufficient time elapses for the formation of a number of impulses, yet the single premature beat stands isolated. And oftentimes the spontaneous premature beat falls in diastole at uncertain intervals from cycle to cycle, or fails to appear for many cycles. The meaning of this haphazard behaviour is usually obscure, but it stands in contrast to the regular incidence of the intrinsic idio-ventricular beat which crops up at calculable instants.

The premature contractions are not the product of rhythmic impulse formation, in the usual meaning of the phrase. It is perfectly true that in exceptional conditions beats, which are presumably of a similar nature, may occur successively and regularly, constituting a newly-developed rhythm. But rhythmicity is not an essential quality of the processes underlying the production of such beats, and even when they occur successively and regularly it is open to question whether rhythmic impulse formation, in its true sense, is responsible for such succession. It is suspected, and with good reason, that the second contraction is actually called forth by the first, the third by the second, and so forth. Regarded in this way, the relationship of the preceding contraction to an idio-ventricular and to a premature ventricular contraction may be stated in the following manner: the preceding contraction signals the birth of the impulse which promotes the idio-ventricular contraction, while it actually initiates the premature beat or promotes its special impulse.

Now while the foregoing discussion is necessarily and admittedly in a hypothetical form, it is of service if it expresses the possibility, nay probability, of an essential chemical distinction between the processes which pave the way to contractions, which may be termed *physiological or homogenetic* and *pathological or heterogenetic* respectively. The former

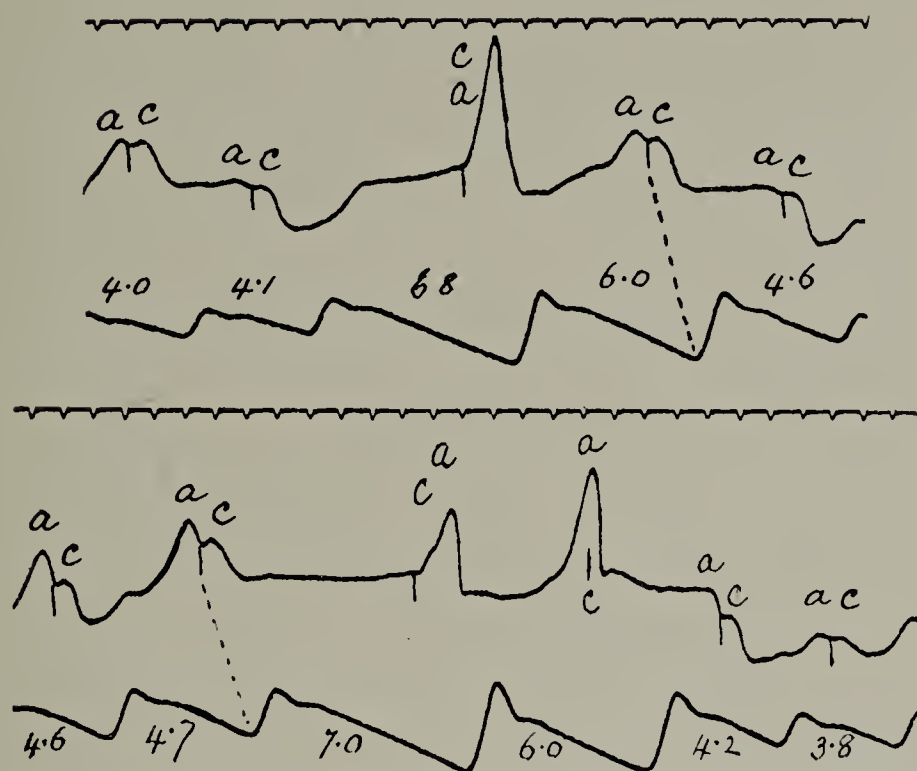


Fig. 87 and 88 (*Quart. Journ. Med.*, 1908-9, II, 358). Polygraphic curves showing escape of the ventricle as a result of sinus slowing. The longest pauses are terminated by spontaneous ventricular contractions, and they coincide with the auricular systoles, producing exaggerated waves in the jugular pulse curve. In this instance the preceding pause varies by about 0.2 sec.*

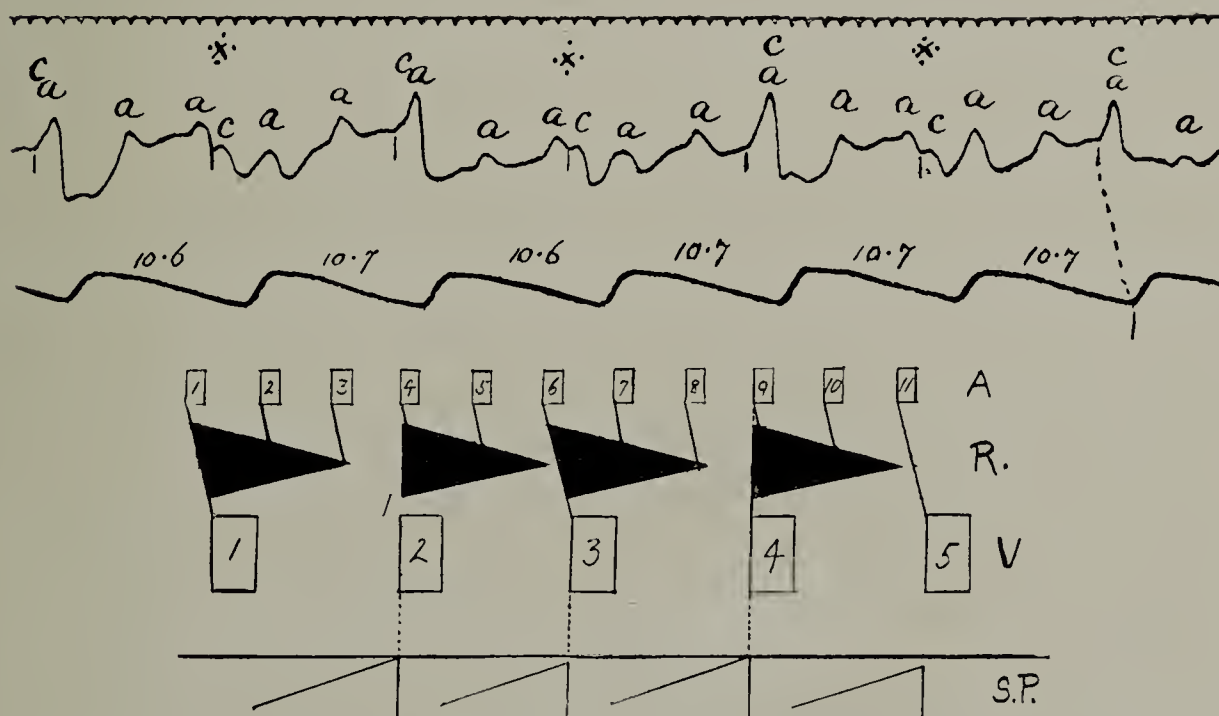


Fig. 89 and 90 (*Quart. Journ. Med.*, 1908-9, II, 361). Polygraphic curve from a case of heart-block. The ventricle escapes at each alternate beat and responds to auricle at each alternate beat. The conditions are such that when the ventricle is about to respond to an alternate auricular impulse (No. 44 and 49 in the diagram, Fig. 90), stimulus production in the ventricle has just reached a sufficient point and anticipates the auricular impulse.

* In many instances it is absolutely constant, see Wenckebach¹¹ and Fig. 77-80.

are specially associated with normal nutritive surroundings; while, as we shall see in the succeeding chapter, the latter arise as a result of gross interferences or under circumstances in which it is believed that normal nutrition is interfered with to a marked extent.

In the case of a ventricular rhythm composed of contractions which appear to belong to the pathological order, the contrast with the idio-ventricular rhythm is one of rate. While the true ventricular rhythm is remarkably slow, the pathological rhythm is rapid; it rarely, if ever, falls below 130 beats per minute, and under special circumstances may exceed 200 per minute in man, and may even surpass 400 per minute in the dog (examples of such rhythms will be found in Chapter XIV). The comparison has been confined to the ventricle, for this muscle area is relatively undisturbed by nervous influences. In the auricle the separation of the homogenetic and heterogenetic rhythm is accomplished with less facility; the identification is not aided in the same degree by a study of rate, for at times the physiological rhythm of the pace-maker may be accelerated to 200; it is accomplished with greater certainty by an examination of the manner in which the rapid heart action has its onset or offset. For example, if the rate of the auricular contractions rises gradually, the new rhythm is probably identical with the old, but its rate has been enhanced; while, if the rate rises suddenly from one cardiac cycle to the next, and the rates before and after the change are constant, it is obvious that the preparatory processes have undergone an abrupt alteration, and the presumption is strong that an entirely new factor has come into play.

The distinction between the two types of cardiac beats is one which the writer believes to be of considerable pathological significance; he is aware at the same time of many difficulties with which the question is beset; and the impossibility of stating in certain specific instances that a rhythm belongs to one or the other category must be admitted. It is possible that a further study of the factors influencing the production of such rhythms and affecting their rates, study which is urgently required at the present time, may afford a basis for a clearer distinction between them.

In the meanwhile, even a partial appreciation of the facts materially assists the interpretation and classification of disorders of the cardiac mechanism.

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CHAPTER XII.

SINGLE HETEROGENETIC HEART CONTRACTIONS.

The premature ventricular contraction.

IN the preceding chapter certain facts were discussed and the reader was impressed with the importance of recognising the significance of premature cardiac contractions as being heterogenetic or of pathological origin. In commencing a more detailed study of our knowledge of the disturbances which result from the presence of beats of this nature, it will be convenient, as hitherto, to take the ventricular premature contraction (often designated ventricular *extra-systole*) as a type. It has been stated that the ventricle is excitable and responds to single induction shocks, of sufficient intensity, applied to it at any moment in its diastole. The magnitude of the contraction which results is independent of the strength of the stimulus awakening it, provided that such an excitation is adequate; a phenomenon discovered by Bowditch¹ and termed the "all or nothing" response. The limits of excitability in the ventricular cycle were first investigated by Edouard Marey,²⁰ and as a result of his observations it became evident that however intense the stimulus, the musculature is refractory during the time of its systole (the *refractory period* of the cycle). An excitation of adequate intensity applied to any portion of the ventricular musculature provokes a series of definite events. The ventricle contracts, and the contraction is propagated from the point of stimulation to the rest of the ventricular tissue.^{6 & 26} Under ordinary circumstances the contraction is confined to the ventricle, and, as a probable consequence, the auricular rhythm rests undisturbed. The events are diagramatised in the accompanying illustration.

The regular beats of the auricle are represented by the narrow black rectangles in the upper line (*A*). The ventricular systoles by the broader rectangles of the lower line (*V*). The premature contraction is represented at *p*, and it is followed by the longest pause which is to be found in the figure. The meaning of the pause and its length will be evident when its time relationships are considered. It was pointed out by Knoll¹³ that the distances between the commencement of the premature contraction and the preceding and succeeding contractions are equivalent,* when added together, to the distance occupied by two whole cycles of the regularly beating ventricles ($a=b$); for an explanation of the phenomenon we are

* A clinical observation.

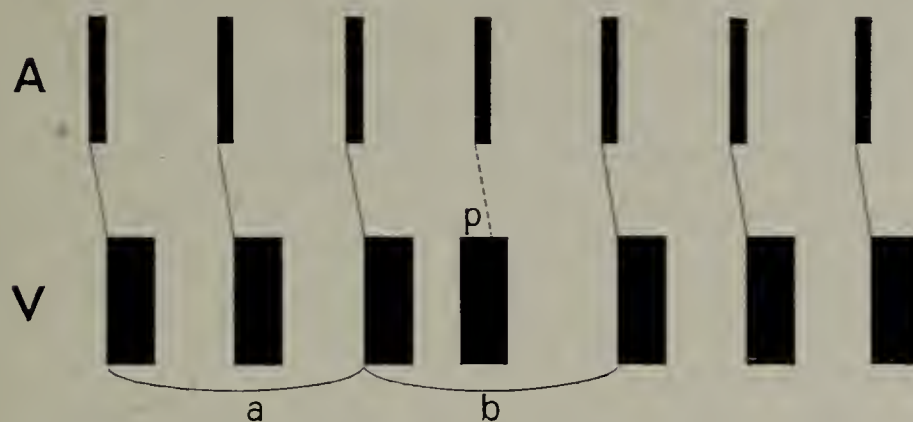


Fig. 91. A diagram illustrating the disturbances which are associated with a single premature contraction of the ventricle (*p*). The auricular rhythm remains undisturbed. The pause following the premature contraction is compensatory, for the distance *a* = the distance *b*.

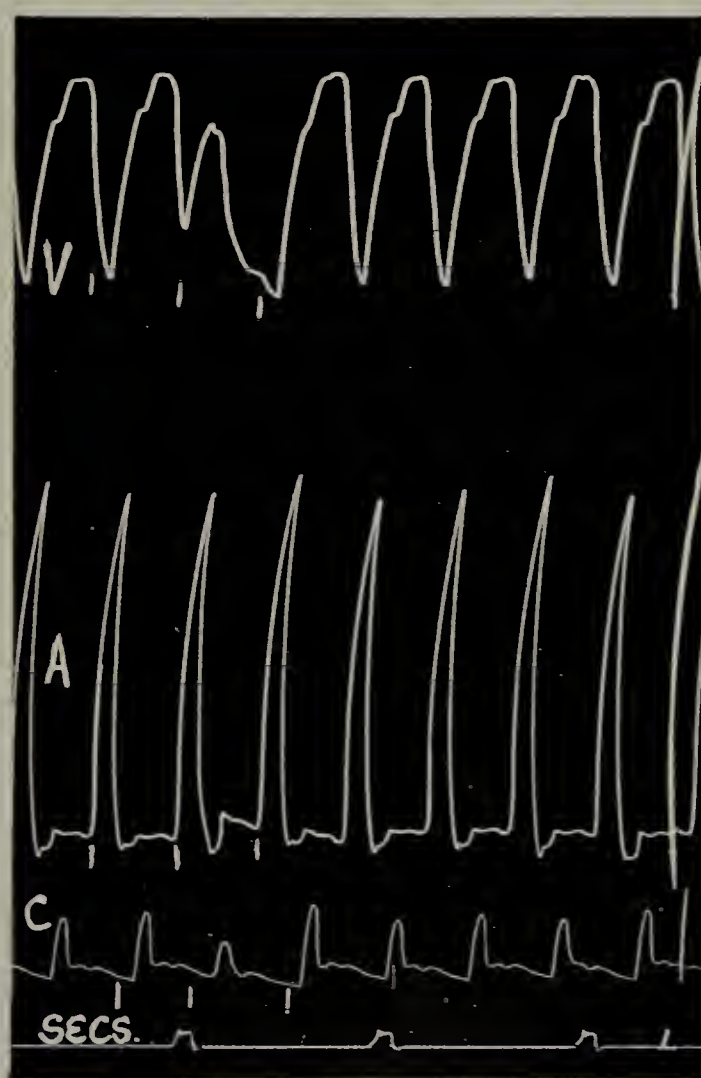


Fig. 92. Myocardiographic curves (*V*=ventricle, *A*=auricle) and Hürthle carotid pressure curve (*C*), from a dog in which the right coronary artery had been tied. Showing a single spontaneous and premature contraction of the ventricle. The pause following the weak beat in the arterial curve is compensatory; the auricular beats maintain their rhythm. The prematurity of the carotid beat is barely perceptible on account of the temporary increase of presphygmie interval. Time in seconds.

indebted to Engelmann, who worked with the frog's heart. It is that the premature or excited contraction has arisen independently of an auricular contraction, and that the succeeding auricular impulse (represented in the diagram by the oblique line) falls upon the ventricle when it is in a refractory state. As a consequence, the ventricle fails to respond to it, and awaits the call of the next auricular impulse. The pause which follows the premature contraction is therefore prolonged, and inasmuch as it makes amends, by its length, for the shortness of the preceding interval, it has been termed the *compensatory pause*. The same phenomena were observed in the mammalian heart by Cushny and Matthews.⁵ An experimental example of a premature ventricular contraction is shown in Fig. 92.

Thus, while the ventricular rhythm is temporarily disturbed, the points at which the following ventricular contractions make their appearance suffer little or no change. The words "little or no change" are chosen deliberately, for usually there is *no* change. But under certain conditions a slight alteration in the relative position of the succeeding beats may occur. It appears as if, with the unusual length of rest, the latency of the ventricular response is decreased, and the systole which succeeds the disturbance appears somewhat earlier than the anticipated point. A lesser degree of dislocation is associated with the second contraction and a recovery of position is complete within one or more cycles. These events are shown in an exaggerated form in the accompanying diagram (Fig. 93).

The absence of response to the regularly placed auricular contraction is attributed, as has been stated, to the refractory condition of the ventricular muscle when it receives the impulse from the upper chamber; but sometimes, when the heart rate is slow and the premature contraction sufficiently early, it may happen that no response is missed, and the premature ventricular contraction becomes a true extra-systole of this chamber (Fig. 94).

This event is referred to by speaking of the additional beat as an interpolation, or as an *interpolated ventricular contraction* (interpolated ventricular extra-systole) (*cp.* Fig. 95).

The premature ventricular contraction is relatively abortive in its power of expelling blood from the heart, and its strength is largely controlled by the position which it occupies in diastole and upon the consequent degree to which the contractile function of the muscle has recuperated: for the recovery is gradual to a point at which the maximal power is restored (Bowditch). The relatively weak expression of the ventricular contraction in the arteriogram (Fig. 92, 96, etc.) is in part the outcome of this lack of fully developed power. It is also produced in certain measure by the small ventricular content at the moment of contraction, and also in some degree possibly by the abnormal course which the contraction wave takes in the muscle which expels the blood. The beat which

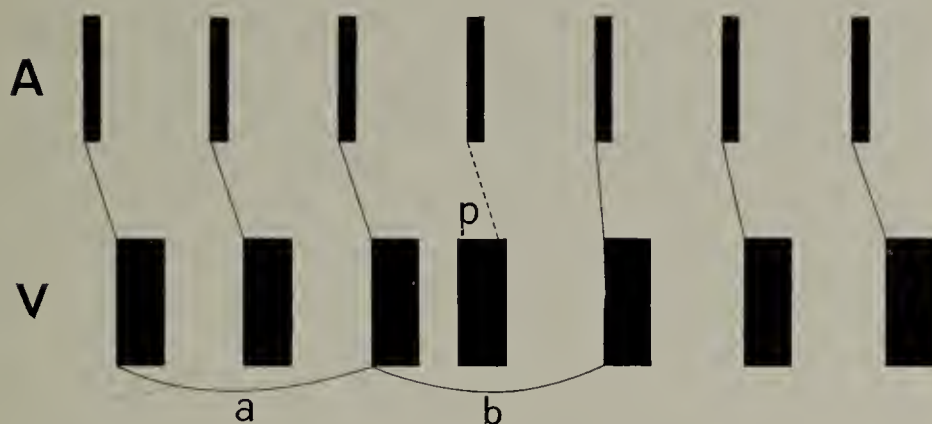


Fig. 93. Similar events to those shown in Fig. 84, but showing disturbance of the spacing of ventricular beats following the premature contraction, as a result of altered conduction.

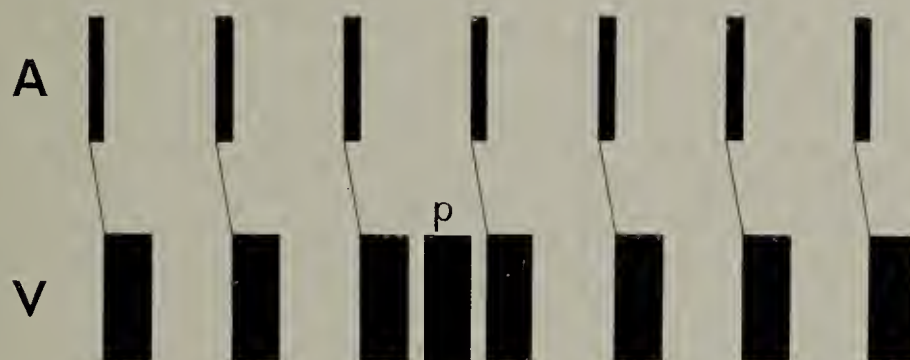


Fig. 94. A diagram illustrating the time relationships of auricular and ventricular contractions when a premature ventricular contraction (*p*) is interpolated.

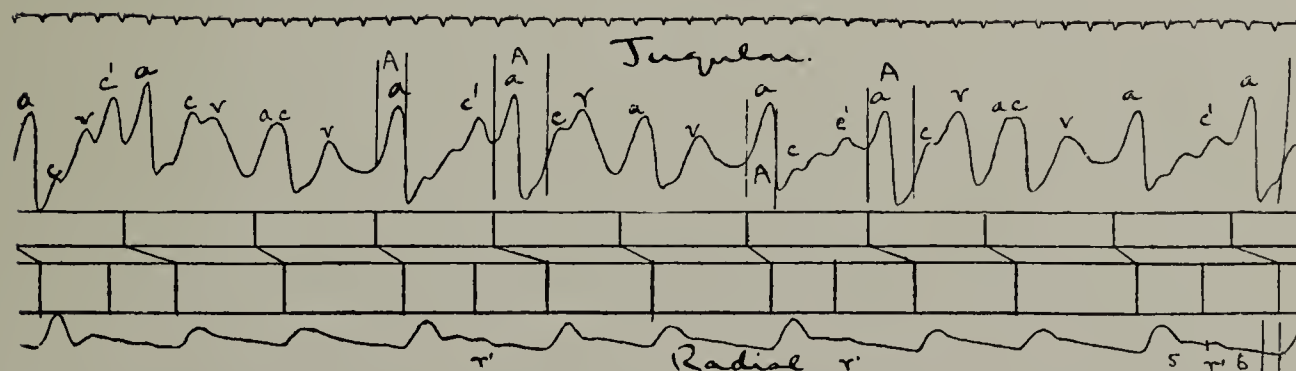


Fig. 95 (Laslett, *Heart*, 1909-10, 1, 84). A polygraphic curve showing regularly occurring interpolated beats arising prematurely in the ventricle. They are marked *r'* in the radial curve. In this instance a considerable increase of the *a-c* interval, succeeding each disturbance, was noted; two pairs of intervals are marked by vertical lines in the figure.

follows the interruption is correspondingly enlarged for reasons the reverse of those which we have considered.²³

By no means infrequently it happens that premature contractions are quite abortive in raising aortic pressure; under the circumstances the beat is no longer recorded in arterial curves (Fig. 97 & 105; the so-called *frustrane* contraction), and, giving rise to the first cardiac sound, is unaccompanied by the second sound which results from the movement and tension of the aortic valves.

The ways in which premature contractions are evoked in the ventricle.

Stimulation of the ventricle, either by mechanical or electric shocks, by the application of heat or crystals of common salt, gives rise to the pathological contractions which we are considering. But they are also produced in other ways. They have been found to result when the pressure in the ventricle is raised abruptly by clamping the aorta.⁹ They occur when the heart muscle is rendered anæmic: the temporary cessation of the inflowing blood following deliberate obstruction of the cavæ is sufficient to produce them²⁷; ligation of the branch of a coronary artery has a similar but more profound effect¹⁵ (Fig. 92, etc.). They are also seen as a result of the injection of certain poisons, and notably digitalis,² adrenalin,¹² aconitine,⁴ muscarine and physostygmine.²⁵ Lastly they occur in disease, and it will be our almost immediate duty to show how they are recognised in man, and to consider the disturbances to which they give rise. But before proceeding to this demonstration, an experimental fact of the highest importance requires recognition. The beats occur under suitable conditions, *when all nervous connections between heart and central nervous system are severed*. This fact is rendered indisputable by their appearance in the isolated and perfused heart. It has also been demonstrated upon the intact heart, which beats *in situ*, and from which the vagal and sympathetic connections have been completely severed (a fact recorded by Hering⁹ and one which the writer has repeatedly confirmed). It has been shown that when the beats occur spontaneously (that is to say, in the absence of mechanical or electrical interference) they do not arise in consequence of impulses reaching the heart through the nerves which supply it; and no experimental damage to the nerves themselves has ever been shown to give direct origin to a contraction of this nature. It is the recognition of these facts which impresses the importance of searching the heart, and not the nervous system, in the patient who is the subject of such disturbances.

The recognition of premature ventricular contractions in clinical subjects.

For the recognition of premature contractions of the heart we are chiefly indebted to the work of Wenckebach¹⁹ and Mackenzie,^{16 & 17} and

it is based upon our knowledge of the time relationships of the several contractions in auricle and ventricle, as they are found in experiments at the time of the disturbance. We deal at present with the premature ventricular contraction.

In the arterial curve the disturbance is seen as an interruption of the regular sequence of contraction of the left ventricle, which may or may not be marked by evidence of the premature beat itself (Fig. 96, 97 and 98), but which shows the characteristic of premature ventricular systoles, namely, the presence of the full compensatory pause and the absence of permanent dislocation of the spacing of the recovered rhythm.

In the cardiogram the early ventricular beat is almost always, in the electrocardiogram always, visible. The venous curve shows the regular and undisturbed sequence of the auricular waves. Where the premature ventricular contraction falls synchronously with the expected auricular contraction, the usual exaggeration of the venous peak is found (Fig. 96). The justification of the interpretation is afforded by experimental curves²² examples of venous curves taken from animals and showing premature ventricular contractions are given in Fig. 97 and 98. The interpretation is fully corroborated by a study of corresponding electrocardiograms, be they clinical or experimental. As in experiment, so in clinical cases, interpolated beats (Fig. 95). are comparatively rare, but as might be anticipated are peculiarly associated with retarded heart rate.

The localisation of the *ventricular* seat of origin of the premature contractions is fully evidenced by arterial and venous curves alone. As we shall see, a further localisation in the walls of the ventricles themselves is now possible, but will be more conveniently considered in the next chapter. In the meanwhile, we may proceed to a consideration of the experimental facts in regard to premature contractions arising at higher levels in the musculature.

The premature auricular contraction.

Many of the experimental observations which apply to the ventricular type of beat, and more particularly the means of eliciting it, are applicable to the contractions which start in the auricle. We shall confine our attention to the differences which exist between them.

An early auricular systole is followed in the normal heart by an early systole of the ventricle which is a response to it (Fig. 99). There is a consequent disturbance in both auricular and ventricular sequence. But under certain conditions the disturbance of rhythm in the ventricle is of slighter grade than is the corresponding arrhythmia in the auricle (b is greater than b'). The difference is brought about by alterations in the rate at which

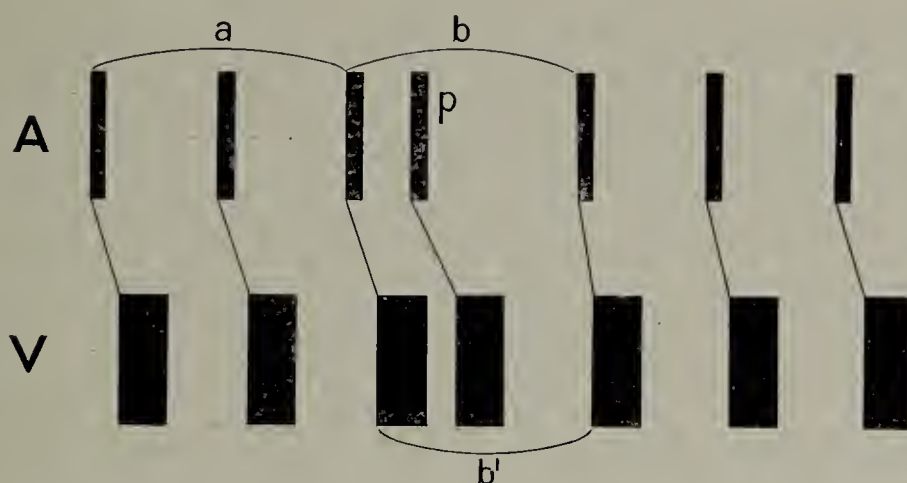


Fig. 99. A diagram showing the time relationships of auricular and ventricular contractions when the sequence is disturbed by a premature auricular contraction. The auricular pause is not compensatory, $a > b$. The occasional alteration of transmission intervals is shown ($b > b'$).

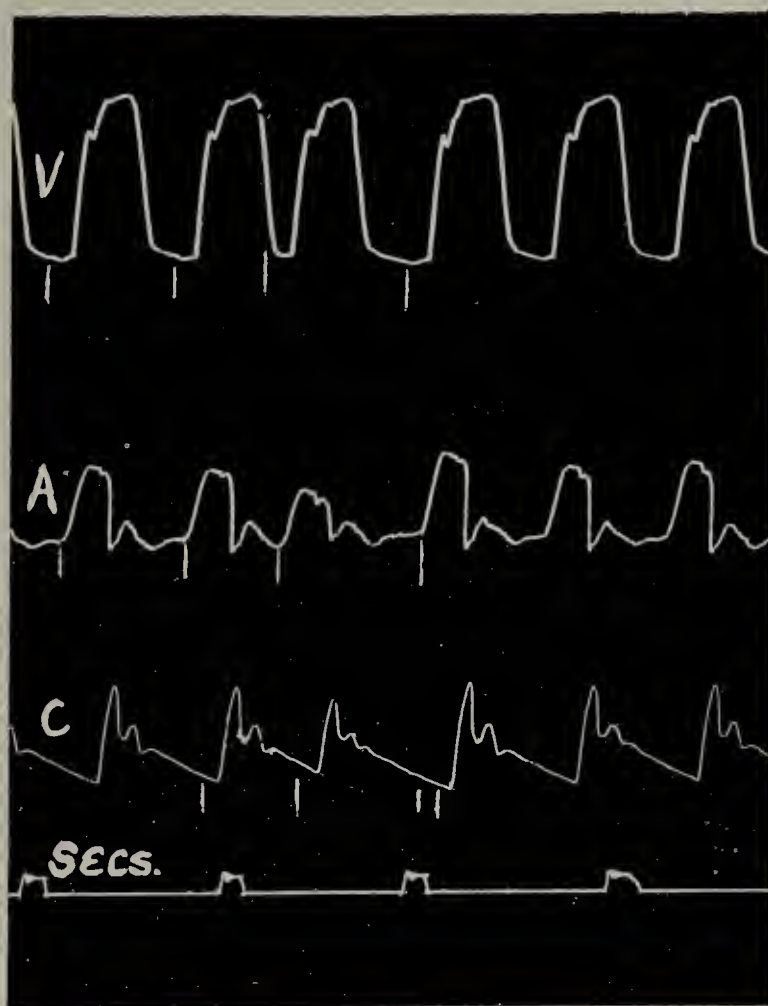


Fig. 100. Myocardiographic curves (A =auricle, V =ventricle) and Hürthle carotid pressure curve (C) from a dog. A single and spontaneous contraction of the auricle is shown; the ventricle follows suit and contracts too early. The pause in the carotid curve is not fully compensatory. A curve taken from an experiment in which the right coronary artery was tied. Time in seconds.

the several impulses are transmitted from one chamber to the other. These events are shown in the accompanying diagram (Fig. 99); it is considered unnecessary to give a similar diagram in which the transmission times are uniform.

✠ The interrupting beat is, as a general rule, short of being compensatory (a is greater than b). It is fully compensatory on rare occasions; but it may be said that the premature auricular contraction is characterised by the relative shortness of the pause which follows it. In fact it was first recognised in man by this very quality. A great deal of time has been spent in the investigation of the length of the pause, and the cause of its variation; but it must be confessed that we are not yet fully conversant with the method of its production. The subject received brief attention in the chapter which dealt with the pace-maker of the heart, and it has been stated that the general findings point to the conclusion that the pause is relatively longer, according as the seat of disturbance moves from pace-maker towards ventricle. The absence of a complete compensatory pause, and the permanent dislocation of the spacing of succeeding systoles is the result of a disturbance of the rhythm of the heart at its spring. The shortening of the pause following a premature auricular contraction in the mammalian heart—it is absent in the amphibian and reptilian heart, where sinus and auricle are clearly demarcated⁶—has been attributed to the retrogression of the wave of contraction to the pace-maker,⁵ and to an extinction of the stimulus material which is in process of formation at this point. But the indications of the rapidity with which the contraction wave passes through cardiac muscle seem to preclude a free admission of this view (the rate of conduction is usually given at 1-3 metres per second), for it seeks to explain, not only the shortening of the pause, but the actual degree of shortening which is present. Thus it is stated that when excitation is applied in the neighbourhood of the great veins (the experiments were performed before the isolation of the pace-maker), the pause is so short as to equal the interval between two beats of the normal rhythm. The formative material is destroyed at its birth-place, so it is said, and commences to reconstruct at its accustomed rate. The majority of the observations certainly favour an acceptance of the fact that the shortest pauses accompany beats produced in the neighbourhood of the great veins; the writer's own experiments upon stimulation of the pace-maker and other auricular areas have yielded facts in general but perhaps not absolute accordance with the hypothesis that the pause is shortest when the excitation falls at the pace-maker. Until there is greater uniformity, we shall not be fully justified in stating that where the pause is equivalent to the interval between two rhythmic beats, the interrupting beat has arisen in the pace-maker. It is probable, however, that it has arisen in its immediate neighbourhood. Premature contractions showing such time relationships have been described as *sinus beats* (sinus extrasystoles).³⁰ The supposed mechanism of a premature contraction of

“sinus” origin is illustrated in Fig. 101, in which c is equal to d . Above the auricular beats, stimulus production in the pace-maker is represented by the triangles (Sp). The full development of an impulse is indicated where an oblique line reaches the horizontal (x, x'); the destruction of formative material is indicated by the sudden drop to the horizontal (y, y') at each auricular contraction.

The difficulties of interpretation are very great where the pause is of such a length that it is greater than the interval between rhythmic beats, and yet when added to the preceding interval it falls short of the length of two full cycles (*i.e.* where the pause fails to be compensatory). Supposing that such a contraction arises in the main mass of auricular tissue, the time which it takes to traverse the distance separating such a point from the pace-maker itself is too minute to account for the increase in length beyond the usual rhythmic length; but at the same time the sinus or basal rhythm is known to have been disturbed. An alternative explanation,⁵ that the premature contraction calls forth the following beat, and that this likewise is premature, is also unsatisfactory. For it is frequently far removed from it in time, and it must be further supposed that the second beat is called forth from the pace-maker, for as shown by electric curves, the type of contraction is of the form of that originating in the pace-maker in all but extremely exceptional instances.*

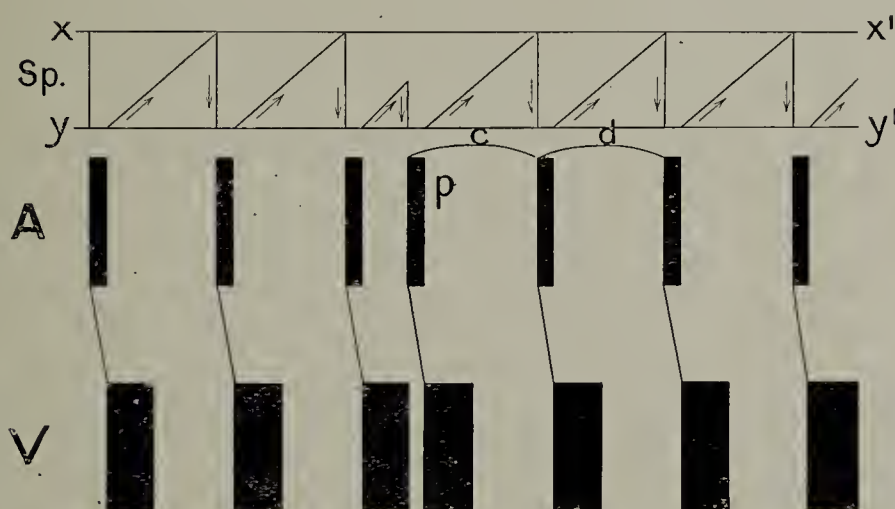


Fig. 101. A diagram illustrating the disturbance of the heart's mechanism when a premature contraction is excited in the neighbourhood of the pace-maker. Sp =stimulus production in the pace-maker; c , the pause following the premature beat, is equal to d the pause following a rhythmic beat.

We must be content for the time being with the observed fact that the premature auricular contraction may be followed by pauses of three lengths: a pause equal to the interval between rhythmic contractions; a pause truly compensatory; or a pause of intermediate length. And it appears certain that if in the case of any premature contraction the pause fails to be compensatory, then such a beat has arisen in the auricle.†

* A single example has come under the notice of the writer, in which the first rhythmic beats following disturbances of a regular rhythm were shown electrically to arise from the same point as the premature auricular contraction itself.

† Always excepting the interpolated contraction.

The recognition of premature auricular beats in man.

The hypothesis of the occurrence in man of premature auricular contractions, originally put forward by Cushny² and Wenckebach,²¹ received full confirmation from Mackenzie's polygraphic records.¹⁵ etc. As in the instance of the ventricular beat, their recognition now rests upon an identification of the instants of onset of the auricular and ventricular systoles over the period of disturbance.

In arteriograms the appearances are the same as those encountered in the case of premature ventricular contractions, with the reservation that in the majority of instances, the compensatory pause fails; that is to say, disturbance of sinus rhythm may be demonstrated. In the phlebograms each ventricular systole is found to succeed an auricular contraction, for each *c* wave is preceded by a wave *a* which can be assigned to no other factor than that of auricular contraction. When the auricular beat is remarkable for its earliness, or when from other cause, such as increased heart rate, the auricular contraction coincides with the preceding ventricular systole, the usual increase in the amplitude of the *a* wave occurs (Fig. 102 and 103). Full justification for the interpretation is obtained from the electrocardiograms in which the events are clearly shown in a single curve (Chapter XIII).

Premature beats arising in the junctional tissues.

A special type of premature contraction, over the exact mechanism of which there has been some controversy, and the precise nature of which is still imperfectly understood, has still to be described. It consists of the simultaneous premature contraction of both auricle and ventricle (Fig. 104). The systoles of auricle and ventricle are more or less coincident. In the diagram (Fig. 104) the contractions are drawn as starting exactly together. This is often the case, but there is considerable latitude; the last phases only of the auricular contraction may coincide with ventricular systole; or on the other hand the auricular systole may be completely buried in the centre of ventricular systole. An example of a spontaneous beat of this nature is shown in Fig. 105 (the myocardiographic levels were working in a reversed direction in this particular experiment). Now the interpretation of beats of this nature turns almost entirely upon the measurement of the intervals between the onsets of the systoles in auricle and ventricle. The pause following the premature contraction may or may not be fully compensatory, and does not help us materially. Both systoles are premature, and as the events are oft repeated it is obvious that either one is dependent upon the other, or that both arise in response

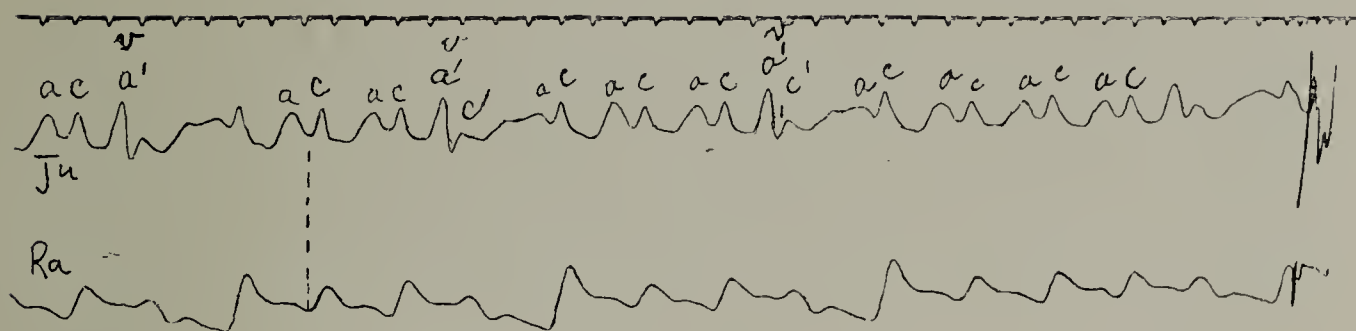


Fig. 102. A polygraphic curve from a patient exhibiting premature auricular contractions. The normal cycles are accompanied by *a*, *c* and *v* waves. The premature radial beats coincide in time with waves *c'*. Preceding the latter are prominent waves due to auricular systoles, *a'*. The auricle contracts prematurely and during the last phase of the preceding ventricular systole.

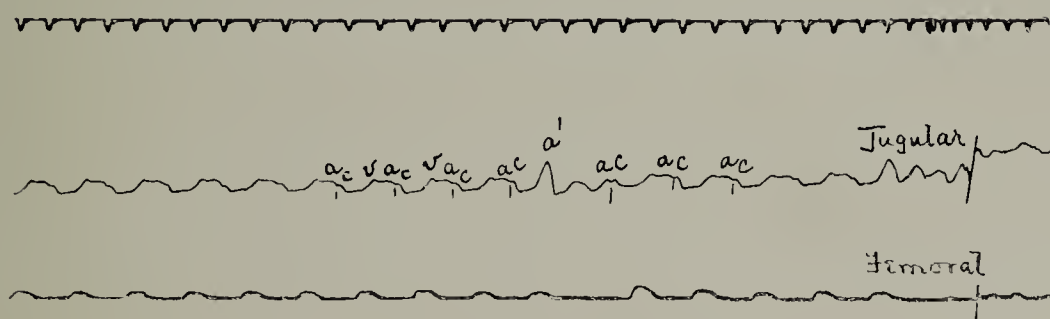


Fig. 103. Venous and femoral curves from a dog, showing the disturbances created by a single premature auricular contraction. The early auricular systole falls within the limits of the preceding ventricular systole, and gives rise to an exaggerated wave *a'*. Synchronism of *a'* and *v*.

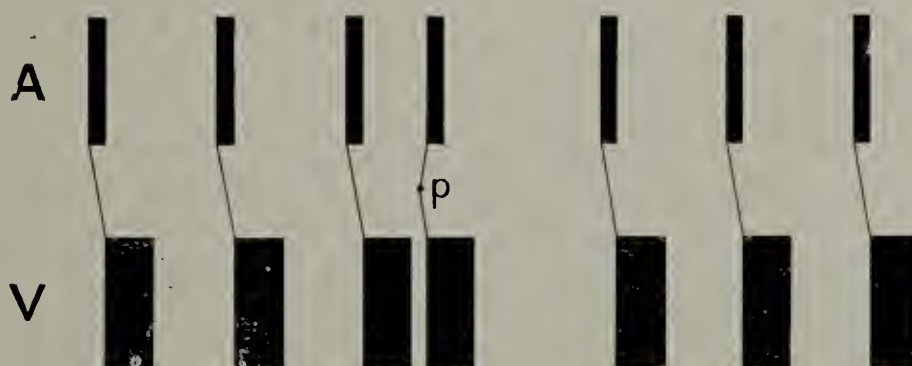


Fig. 104. A diagram illustrating the mechanism of the heart when the sequence is disturbed by a premature nodal beat.

to a common cause. The space between the onsets may be present as an *As-Vs** or as a *Vs-As*† interval, according to whether auricle or ventricle contracts first; if the beats are absolutely synchronous in onset, the interval is necessarily absent. If an *As-Vs* interval is present, it is shortened (otherwise the cycle as a whole would fall into the category of true premature auricular contractions). The shortening of *As-Vs* is evidently a necessary accompaniment of the coincidence of the contractions; and on account of this shortening it is held that the ventricle does not contract in response to an impulse derived from the main mass of auricular tissue. In all instances where a shortened *As-Vs* interval exists as an accompaniment of regularly occurring premature contractions, the simultaneous contraction in the two chambers is assigned to impulse formation in the junctional tissues. The accuracy of interpretation obviously depends in an individual instance upon the delicacy of the measurements of the onsets of the systoles, and upon our knowledge of the transmission rates of impulses travelling up or down the junctional tissues. In experimental work the former presents little difficulty, in clinical work the difficulties are sometimes great. In regard to the transmission rates, we have certain important evidence. The transmission of an impulse, starting in the ventricle and travelling to the auricle, is less rapid than is the transmission of an impulse starting in the auricle and proceeding to the ventricle. In the dog the intervals *As-Vs* and *Vs-As* where ventricle responds to auricle or auricle to ventricle respectively, are approximately .1 and .14 sec.¹⁵ It follows as a consequence that where a premature beat is accompanied by a *Vs-As* interval (as in Fig. 105) the auricle cannot be held to have responded to ventricle unless the *Vs-As* interval in question exceeds the *As-Vs* interval of the rhythmic beats, in the same animal.

The last-named relationship of the instants of onset of *As* and *Vs* in a single premature beat are only met with on rare occasions (an experimental example is given in Fig. 106, and the phenomenon, "retrogression" of the heart beat, is more fully described in Chapter XIV). Thus we proceed to the conclusion that where there is coincidence of *As* and *Vs*, the premature contraction in one or other chamber is the result of a single impulse formed between them, *i.e.* in the junctional tissues, if an *As-Vs* interval is present and is curtailed; and if a *Vs-As* interval is present, which does not exceed the normal *As-Vs* interval in length, the same conclusion holds good. There is consequently a wide variation of intervals over which premature contractions may be assigned to impulse production in the junctional tissues. Is a further localisation possible? The answer to this question depends upon the acceptance or otherwise of Hering's recent observations.¹⁰ This investigator concludes that the greater part of the transmission time from auricle to ventricle or ventricle

* The interval separating the commencement of an auricular and the succeeding ventricular contraction.

† The interval separating the commencement of a ventricular and the succeeding auricular contraction.



Fig. 105. Inverted myocardiographic curves (*V*=ventricle, *A*=auricle) and Hürthle carotid pressure curve (*C*) from a dog, in which the right coronary artery had been tied. The figure shows a single spontaneous and premature contraction of both auricle and ventricle (nodal beat). The pause is not quite fully compensatory; no trace of the premature beat is seen in the arteriogram. Time in seconds.



Fig. 106 (*Heart*, 1909-10, 1, 120). Myocardiographic curves (*V*=ventricle and *A*=auricle) and Hürthle carotid pressure curve (*C*) from a dog, in which the right coronary artery had been tied. The figure shows a bigeminy as a result of premature ventricular contractions retrograde to the auricle. The interval between auricular systole and ventricular systole of a normal beat is considerably shorter than that between ventricular systole and auricular systole of the abnormal cycle. (Taken subsequent to the occurrence of frequent retrogression.) Time in seconds.

to auricle is lost in the auriculo-ventricular node. If his measurements are correct, it follows that all the beats showing a variation from a shortened *As-Vs* interval to a *Vs-As* interval, provided that the latter is not excessive, arise in the node.

The recognition of premature junctional beats in man.

The difficulty of recognising these beats in man, where it exists, lies in the accurate fixation of intervals, for in polygraphic curves the *a* and *c* waves are combined. Where *a* and *c* can be shown to fall absolutely together, or where the *a-c* interval is merely curtailed, they may be identified.^{18 & 19} An example of such a beat is given in Fig. 107. But where the point of onset of *a* is indefinite, and a *c-a* interval is suspected, it may be impossible to exclude the possibility of retrogression. This interpretation has indeed been put forward by Volhard²⁸ and others. But our knowledge of the rarity of single "retrograde" beats in experiment renders the interpretation extremely questionable.

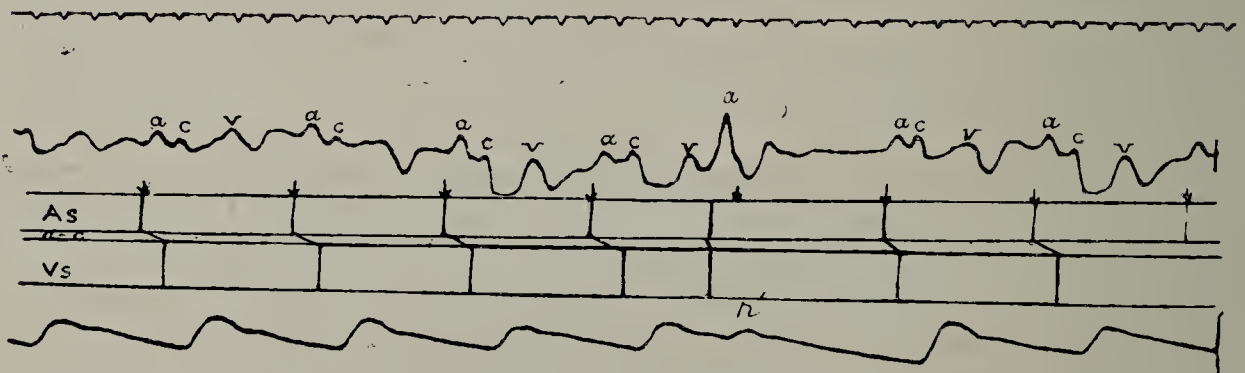


Fig. 107 (*Mackenzie Quart. Journ. Med.*, 1907-8, 1, 142, Fig. 16). A polygraphic curve showing a single premature contraction of nodal origin. *a*¹ and *c*¹ fall together and produce an exaggerated wave.

The measurements of the intervals and identification of premature contractions of junctional origin are fully corroborated by the electric curves, which will receive further consideration.

When premature contractions replace each second rhythmic ventricular contraction, *pulsus bigeminus* appears (Fig. 106), or the pulse rate is reduced to one-half, according as the premature ventricular beats are or are not transmitted to the wrist. If the premature contractions replace each third rhythmic beat, the pulse may show *trigeminus* or *bigeminus* according as such beats are or are not transmitted to the wrist. The heart sounds and murmurs accompanying occurring premature contractions, regular or otherwise, depend upon the chamber in which the beats originate and upon the strength of the premature beats. A knowledge of their nature explains many groupings of heart sounds which are incomprehensible to those who are unacquainted with the findings of graphic records.

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CHAPTER XIII.

SINGLE HETEROGENETIC HEART CONTRACTIONS (*continued*).

The electric changes accompanying premature auricular contractions.

STRESS has been laid upon the heterogenetic quality of premature cardiac contractions, in contrasting them with the homogenetic contractions of the regular sinus rhythm. A further and equally important character pertaining to such beats remains for discussion; they are for the most part *ectopic* in origin.^{5 & 6} That is to say, *they arise in the majority of instances, not at the pace-maker, but in some other portion of the cardiac musculature.* Now this is obviously the case where the premature ventricular contraction is concerned, but it was not so evident in instances of premature auricular contractions, before such beats were recorded electrocardiographically.

In the chapter in which the site of the pace-maker is discussed, the auricular electric complexes resulting from stimulation of various areas of the auricle are referred to. In a given auricle no two points of stimulation yield precisely the same type of auricular complex, though the more nearly the points of excitation approach each other, the greater is the resemblance between the curves obtained. The complexes yielded by stimulation of the superior vena cava area are of similar general conformation to those of normal heart beats in the same animal. The auricle gives rise to an electric change, which chiefly consists in base-apex leads of a variation in the upward or base-negative direction. While the identification of various forms of auricular complex with certain areas of the auricle is by no means concluded, certain broad rules may be substantiated. The auricle, speaking of right and left auricles as a whole, may be divided into three areas, an upper, a middle and a lower zone. Stimulation of the upper zone will yield complexes of a chiefly upright (or base-negative) form, excitation of the lower zone those of a chiefly inverted (or apex-negative) form, while stimulation of the central zone will yield curves which are in the main horizontal (isoelectric).⁶ When those patients who exhibit premature auricular contractions are examined electrocardiographically, a striking fact is at once revealed. Although it may be impossible to locate the actual seat of origin of the heterogenetic contractions with any degree of accuracy, yet the contractions usually give rise to auricular complexes of anomalous form.⁶ The complexes have outlines which diverge in more or less marked degree from those which accompany normal and sequential

contractions (Fig. 49, 111, 112, 113, etc.). Thus it becomes evident, that the majority of the premature auricular contractions which are encountered clinically are ectopic in origin; they arise at points other than the pace-maker. While it cannot be denied that the seat of impulse formation may not coincide on occasion with that of the pace-maker, yet it must be acknowledged that such syntopism is of rare occurrence; the origin of a spontaneous premature beat at or near the pace-maker is infrequent, but it is not known to be more or less infrequent than would be expected from a comparison of the size of the pace-maker with the extent of the remaining auricular tissue.

The ventricular complex which arises as the result of a premature auricular contraction.

We have seen that a premature contraction of the auricle is usually responded to by the ventricle. Where the transmission of impulses from upper to lower chamber is impeded the response may fail (Fig. 49).

We deal at the present time with the appearances of the ventricular complexes, where response has occurred. The impulse propagated by a premature auricular contraction enters the single path which is open to it, the *A-V* bundle, and spreads through the two main divisions of the bundle to their arborisations upon the inner surfaces of the ventricles. It enters the ventricle through its usual field of reception. The ventricle as a whole is excited by an impulse of supraventricular origin. When the heart is in a normal state, an impulse of this kind gives rise to the expected electric picture, that which accompanies the normal heart beat. It consists of the usual variations, *R* and *T*, with perhaps traces of *Q* and *S*, according to the outline of the normal contraction for the individual heart studied (Fig. 108 and 109).

At times the supraventricular impulse awakens a ventricular contraction of an unusual form; so far as our present knowledge leads us, it would appear that it does so, when conduction is demonstrably impaired in some part of the musculature.^{5 & 6} And the observation that, when transmission is interfered with, an anomalous ventricular complex may result, is to be anticipated *a priori*, for in the event of the damage being located in a single branch or in a part of the arborisation of the bundle, then the usual distribution of the impulse to the ventricular walls must undergo change. The alteration of the complex when a single main branch of the bundle has been cut has been proved in fact by Eppinger and Rothberger's experiment (cp. Chapter III).

The variations in ventricular complexes following premature beats arising in the auricle are numerous, and it will be sufficient to notice the

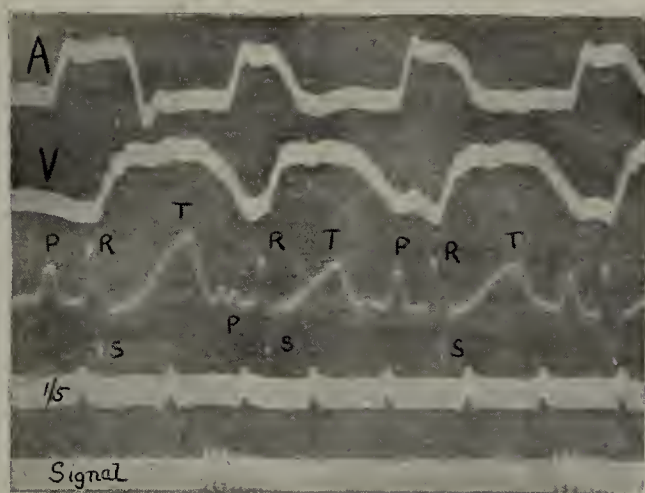


Fig. 108. Myocardiographic curves (*A*=auricle, *V*=ventricle) and electrocardiogram from a dog. A single premature auricular contraction, excited by means of an induction shock applied to the right auricular appendix (at its base and 1.5 cm. from the sulcus) is shown. The excitation is signalled in the lowest line. The first cycle in the electrocardiogram shows *P*, *R*, *S* and *T* variations. The second cycle is premature. It consists of a ventricular complex (*R*, *S* and *T*) of perfectly normal outline; it is preceded by an auricular complex of anomalous type (bifurcated in the centre). The cycles which follow belong to the normal rhythm.

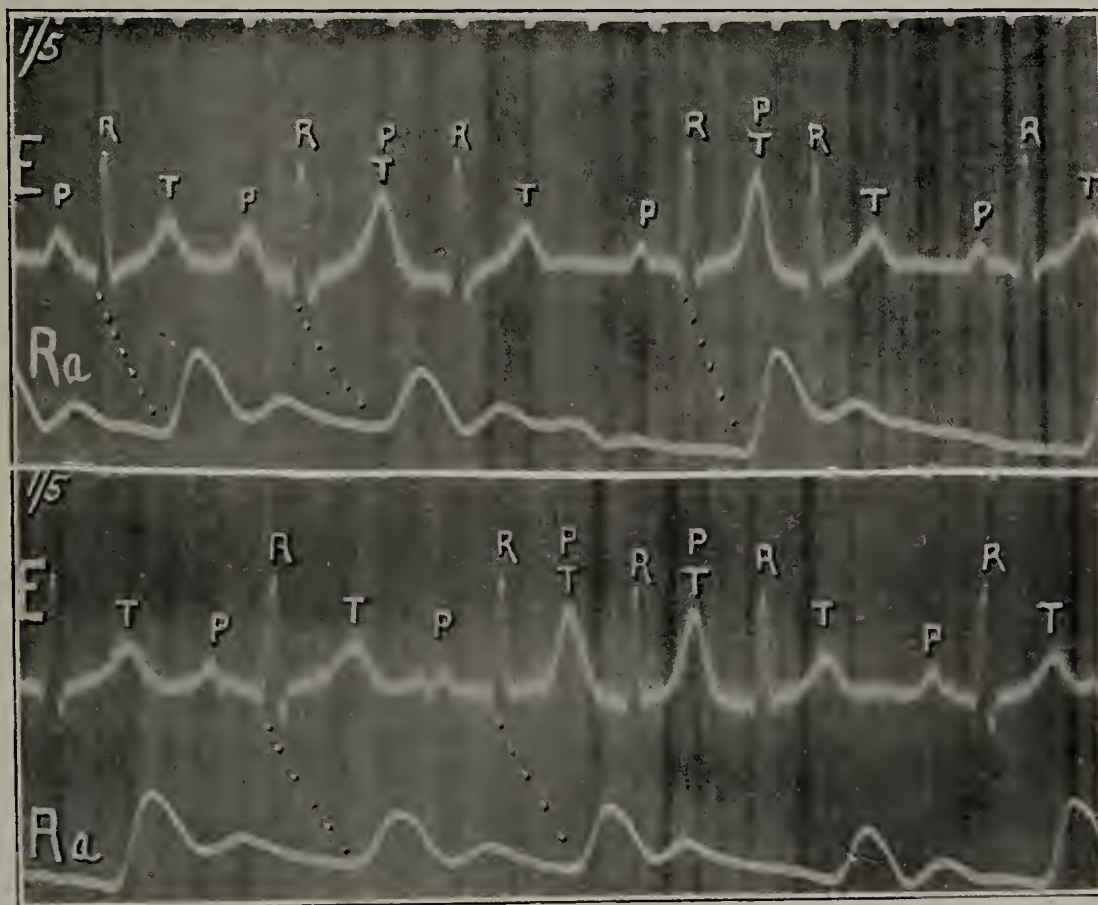


Fig. 109 (*Hart*, 1909-10, I, 306, *Fig. 12*). Electrocardiographic and radial curves from a case showing premature auricular contractions. Two are shown in the upper curve; each is accompanied by a ventricular complex of normal outline. Each is preceded by an auricular complex which coincides with the preceding *T* variation, giving rise to a large composite curve P_T . In the second strip two premature contractions follow each other. The electric appearances are similar. The first premature beat fails to affect the radial pulse curve.

chief variations which are known, and more especially those which may be confounded with complexes associated with ectopic beats of ventricular origin. Relatively small variations are frequent. A decrease in the base-negativity of the peak *T* may be encountered; and in exaggerated instances, *T* may be converted from an upright to an inverted type of curve; the change is usually accompanied by a lesser degree of

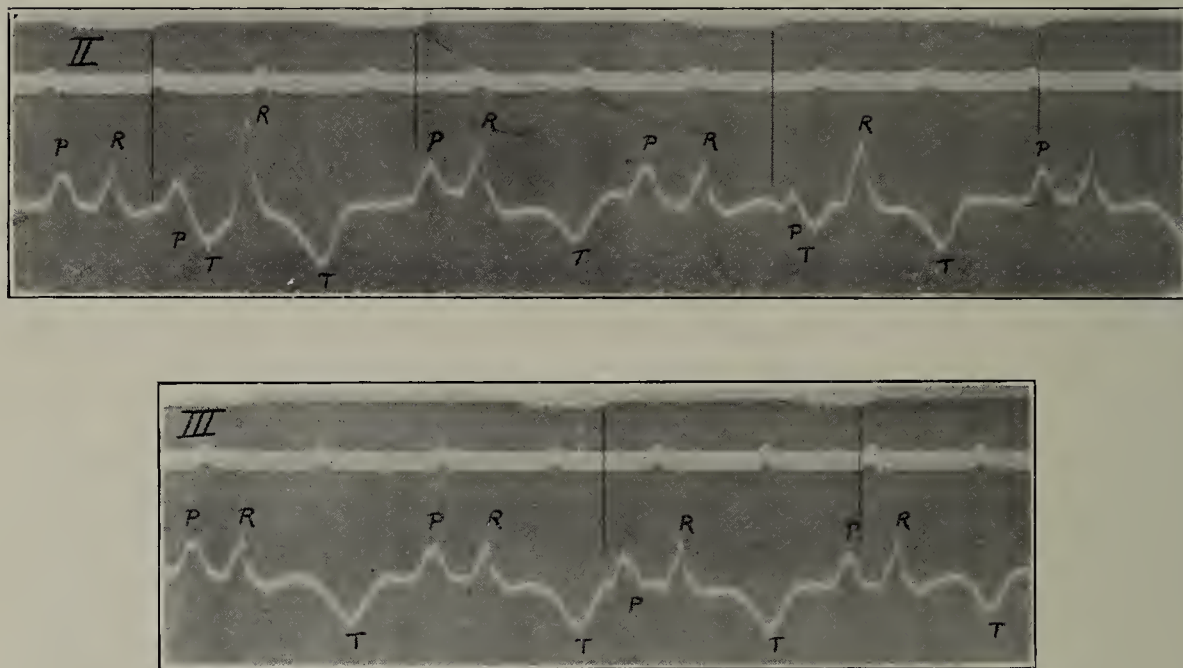


Fig. 110 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 23, *Fig. 7*). Two electrocardiograms from a dog, showing premature contractions, arising as a result of stimulation of the area of the pace-maker. Three are shown (two in the first and one in the second strip). The ventricular complexes vary according to their prematurity, *R* is increased in the upward, *T* is increased in the opposite direction. The auricular complexes have the same outline as those of the normal beats.

alteration of *R*, and of *S* when the latter is present. Secondly, *R* may show a great increased amplitude, and this change is often associated with an inversion of *T*. A clinical example of this nature was discussed in a previous chapter (*Fig. 47*). A somewhat similar experimental instance is shown in *Fig. 110*. The peak *R* of a supraventricular beat is always of relatively short duration. The first base-negative phase of a beat arising in the base of the right ventricle (*Fig. 48*) is relatively much longer. Thirdly, there may be a notable increase of *S*, with or without changes in *R*. In its fully developed form this third type of complex diverges greatly from the normal type, but transitional forms are encountered (*Fig. 112*). It is met with, both clinically (*Fig. 112* and *113*) and experimentally (*Fig. 111*). Other and even more varied forms occur at times (*Fig. 113* and *114*). An acquaintanceship with the several *aberrant* types of complex, as they may be termed,

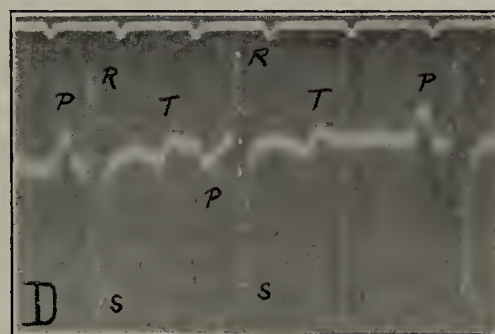
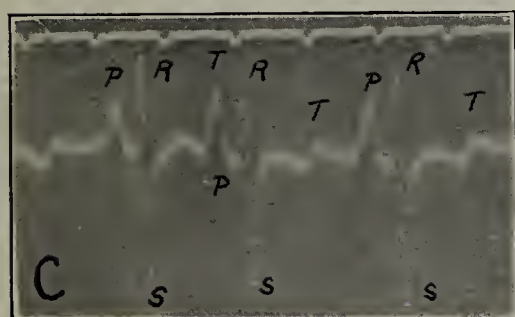
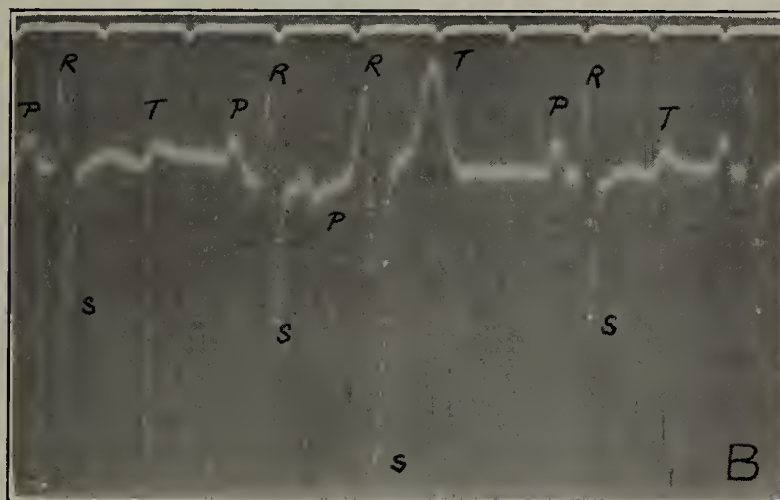
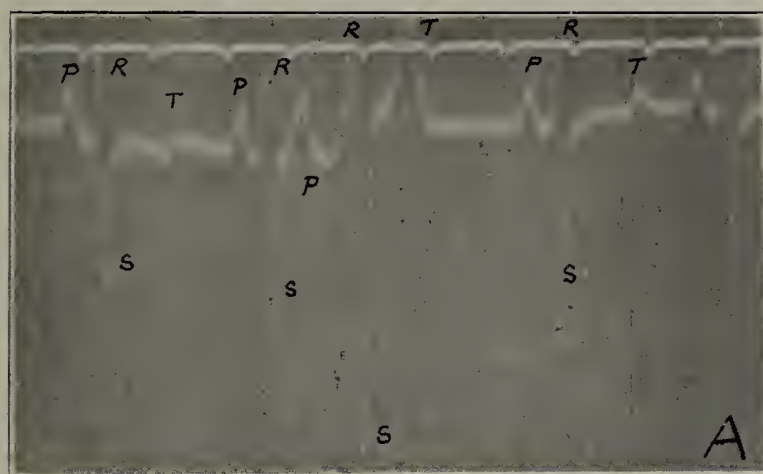


Fig. 111 ($\times \frac{2}{3}$) (*Heart*, 1910-11, II, 23, *Fig. 15*). Four experimental curves. Each shows a premature auricular contraction. *A* and *C* were excited from the superior, *B* and *D* from the inferior caval area. In *A* and *B* the beats occur early, in *C* and *D* later in diastole. With the earliest beats there is marked exaggeration of *S*. The auricular complexes vary according to the point at which the stimulus was applied.

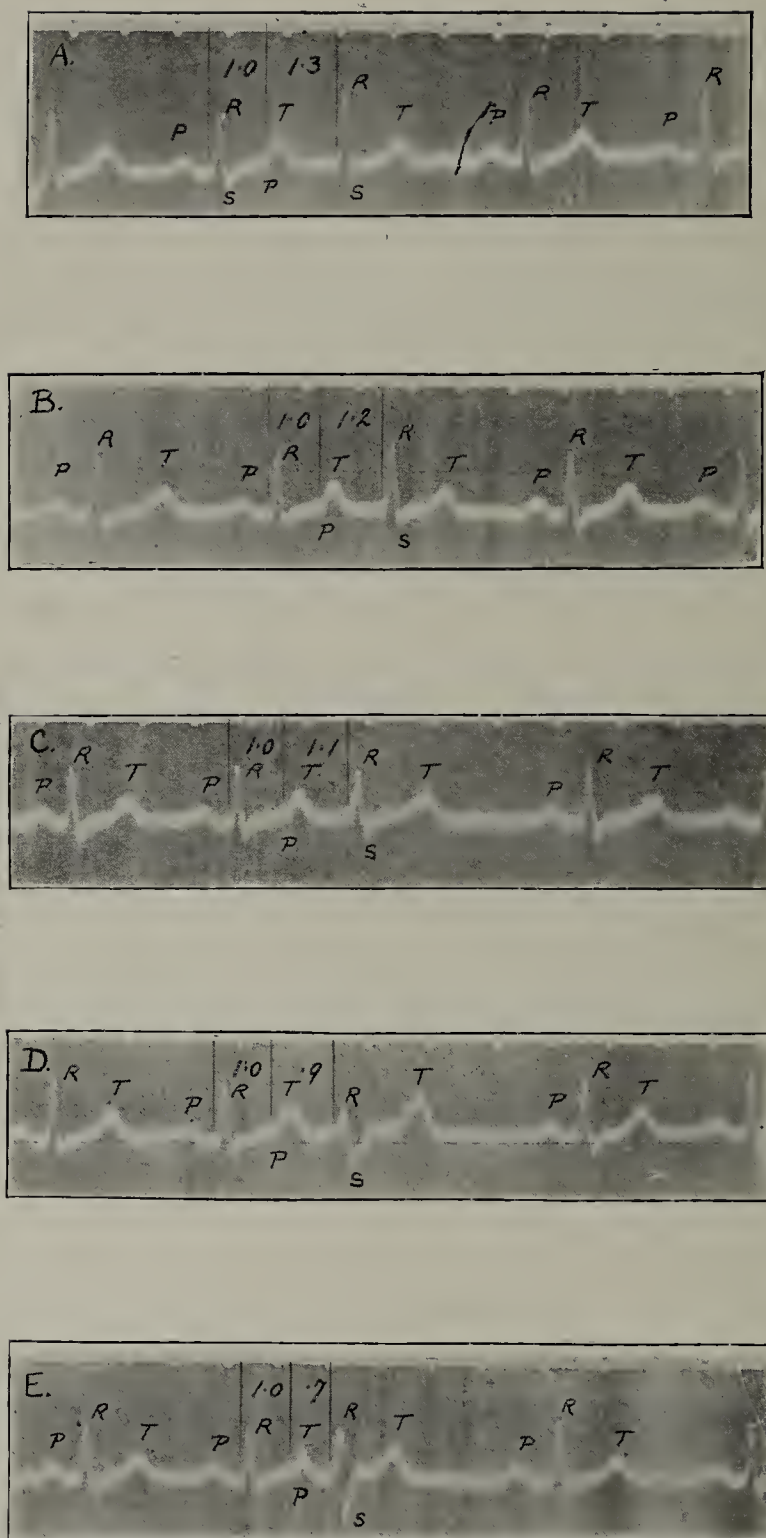


Fig. 112 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 23, *Fig. 10*). Five curves from one case. Each shows a single premature auricular contraction. The auricular complexes are inverted and fore-shorten the initial phase of the preceding *T*. The ventricular complexes show considerable variation, according to whether they stand late or early in the diastole. The curves are arranged to show a gradual transition of the types. There is an accompanying transition of *P-R* intervals which accounts for the positions in which the premature beats stand. The chief change is an increase in the amplitude of *S* (curve *E*). For comparison with the last figure. Fig. 49 and 113 are from the same case.

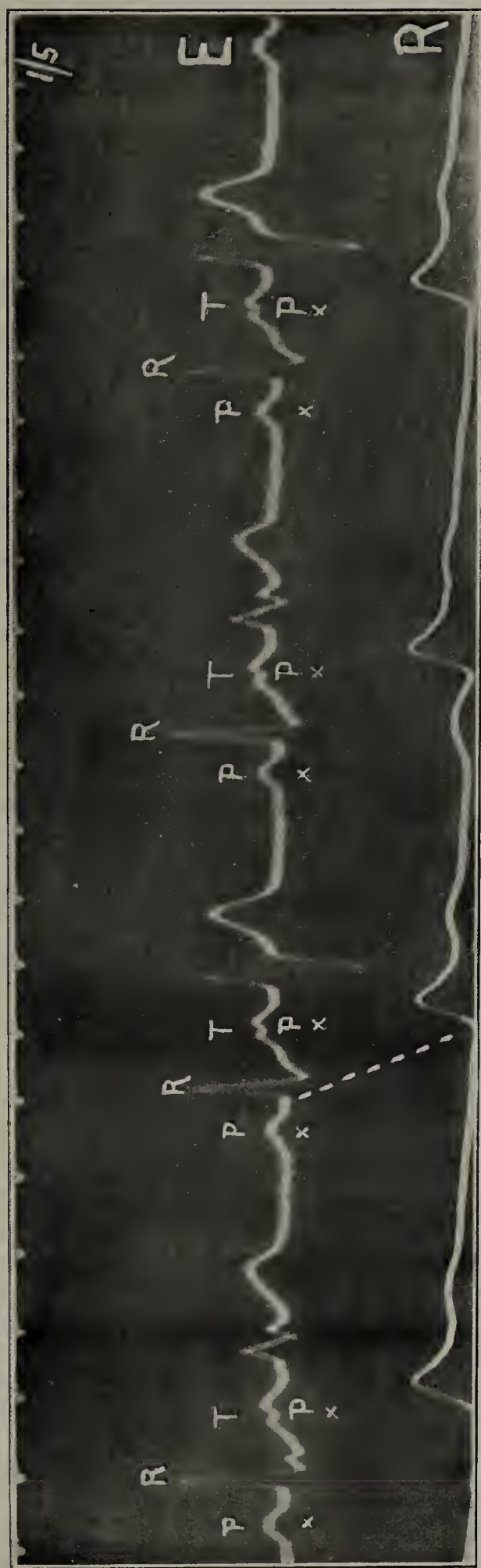


Fig. 113 (*Heart*, 1909-10, I, 279, Fig. 17). Electrocardiogram and radial curves from the same case, showing premature auricular contractions. The anomalous *auricular* complexes are alike (compare Fig. 175), showing that they arise within a limited area. The ventricular complexes are of two types, and they fall alternately. The type with deep S variation has been described already. The second type is also a characteristic aberrant ventricular complex of supraventricular origin. It has been described by Kraus and Nicolai³ as associated with "nodal" beats, but the impulse which gives rise to it usually arises in the main mass of auricular muscle.

is essential. There is danger in examining the curves pictorially; careful measurement, the identification of each auricular and ventricular representative in the curve as a whole, and the careful plotting of such curves are of great importance. Nay more, it is oftentimes imperative to examine the evidence derived from all sources, electric and polygraphic, and to institute a close comparison between them.

The electric waves accompanying premature beats, in which auricle and ventricle contract together.

In regard to beats arising in the junctional tissues, our present knowledge is confined to those in which the *As-Vs* interval is present but reduced. The ventricular complex is of normal outline, the auricular complex is inverted, indicating contraction from below upwards (as opposed to the normal course, from above downwards), and the *P-R* interval is reduced. A single example of such a beat is shown in Plate II, Fig. 218, which will receive closer attention in a later chapter.*

The electric curves given by premature contractions arising in the ventricle.

Just as the *auricular* complexes accompanying beats which arise ectopically in the auricle are of anomalous form, so also are the *ventricular* complexes which are associated with ectopic ventricular beats. And as the ventricular musculature is far more extensive than the auricular, so the types of anomalous ventricular complex are more numerous. Two experimental examples of curves resulting from excitation of the ventricular muscle were published by Kraus and Nicolai.^{1 & 2} In a recent publication,³ they recognise an additional type;³ three types in all. The general statement of these authors is substantially correct. A contraction of the dog's ventricle excited from the apex gives rise to a diphasic curve, of which the first phase is directed downwards (apex-negativity), and the complex as a whole is an indication of the general direction of flow of the contraction wave from apex to base (Fig. 14 & 123). On the other hand a contraction excited from the basal portion of the heart elicits a diphasic variation of the opposite type, indicating first base-negativity and later apex-negativity (Fig. 14 and 117 *I V*). If points of stimulation are tested which lie between base and apex the primary and upward deviation of the basal type of beat gradually diminishes in amplitude and a point is eventually reached in travelling towards the apex at which the resultant

* A special type of curve (similar to the first and third aborrant complexes shown in Fig. 113) has been described by Kraus and Nicolai³ as the accompaniment of "atrioventricular" premature beats. But no evidence is brought forward for this co-relationship. The type of curve may be found when premature auricular contractions are present, as we have seen.

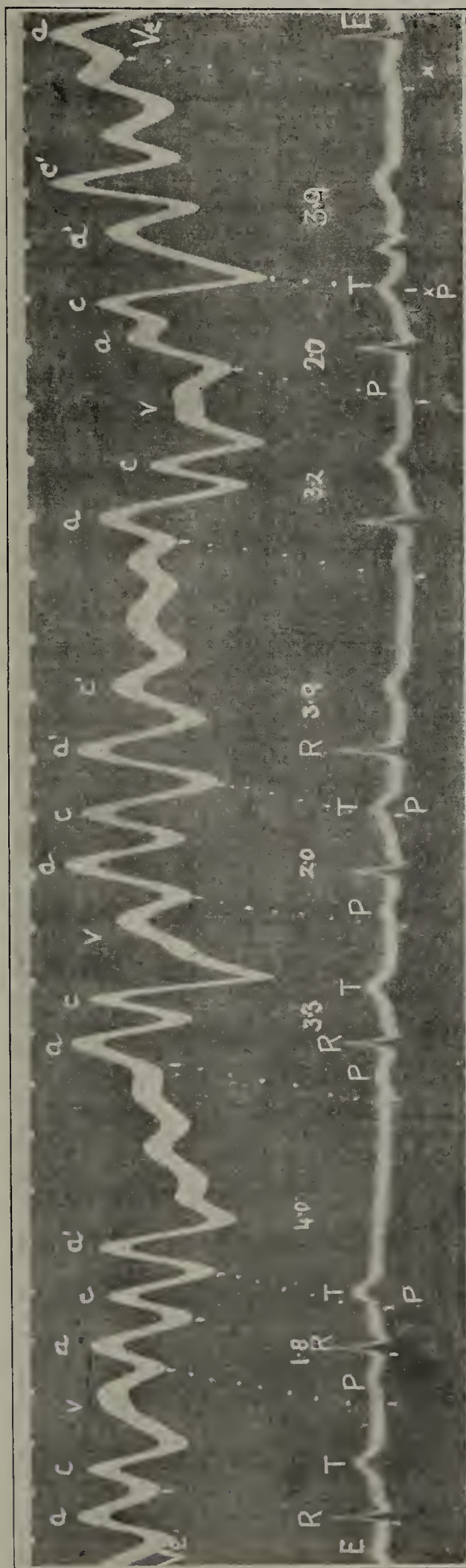


Fig. 114 (*Heart*, 1909-10, I, 273). Venous and electrocardiographic curves from the same case. Several types of ventricular complex, associated with premature auricular contractions are shown. The first anomalous *P*, which falls as usual with the preceding *T* and notches it in the downward direction, is blocked. Its presence is indicated by the wave *a'* in the phlebogram. Two other premature beats each accompanied by *a'* waves are seen; they are associated, the first with the usual, and the second with an aberrant type of ventricular complex. Several curves are given from a single case because it has been fully studied and because the comparison of the complexes is more readily accomplished. But the types described have been met with in a number of patients.

electric curves are of small amplitude and in which there are slight variations about the isoelectric position (Fig. 130 *III*). As the auricle may be roughly divided into three zones, so may the ventricle, an upper, a central and a lower. Of the ventricular zones the upper and lower yield, on excitation, curves of which the phases are of great amplitude, and of which the initial phase is directed upwards in the one instance, downwards in the other (Fig. 14). In their original account, Kraus and Nicolai^{1 & 2} stated that these two types represented contractions initiated in right and left ventricle respectively (for on the front of the heart the right ventricle occupies a basal and the left an apical position), and they concluded that they originate in a complete or partial hemisystole of the heart. They were of opinion that the contraction initiated in the right ventricle was in the main confined to this ventricle; that the normal electrocardiogram is a composite of the two curves, left and right, and that the current produced by stimulation of one or other ventricle is unbalanced, or but partially balanced by contraction of the opposite chamber. Now although there is no evidence of the occurrence of hemisystole in the mammalian heart (it presupposes the possibility of heart-block in the ventricular walls), the proposition of Kraus and Nicolai has been freely quoted as evidence of hemisystole. It is quite untenable, as originally stated, and has recently undergone considerable modification at the hands of these authors themselves.³

The facts in regard to premature ventricular beats are those which might be anticipated. No two points of stimulation yield precisely the same resultant curve (Fig. 130), and an infinite variety of curves may be obtained from one and the same heart. Nevertheless the majority of curves conform in the main to one of the three types instanced by Kraus and Nicolai. The type of curve usually recognised as apical may be obtained in modified forms from a large area of the musculature, which often extends on the dorsal surface of the heart almost to the auriculo-ventricular groove, an area extending so far towards the base that it is difficult to understand why the first deviation is strongly in the apex-negative direction (Fig. 116). Satisfactory explanations of the types of curve originating in definite areas are often extremely difficult to find, and it is probable that in the future closer attention will have to be paid to the arrangement of the ventricular muscle fibres, the directions in which they run, and the points at which the several layers unite.

There are many points of interest in regard to these anomalous ventricular complexes, and some of them are of importance in the analysis of disordered cardiac contraction.

The length of an anomalous ventricular complex is equal within small errors of measurement to that of the normal ventricular complex of the same animal⁵ (Fig. 118 etc.). And this rule, to which but few exceptions have been found in scores of observations, seems to hold good at whatever



Fig. 115 ($\times \frac{5.6}{8.0}$). An electrocardiogram from a patient, showing premature ventricular contractions. There are two single beats and a pair of beats of this nature. The anomalous complexes all have the same outline; there are slight modifications according to their relationships to the rhythmic P summits, which are usually superimposed upon them.



Fig. 116 ($\times \frac{5.5}{8.0}$). An electrocardiogram from a dog; single induction shocks (make and break) were thrown into the base of the left ventricle (high up on the dorsal surface and near the intraventricular groove); the chest wall was intact. The points at which stimulation occurred are shown by means of small arrows in the figure. Where the excitations fall in the refractory periods there is no response. The figure is given for comparison with the clinical curve.

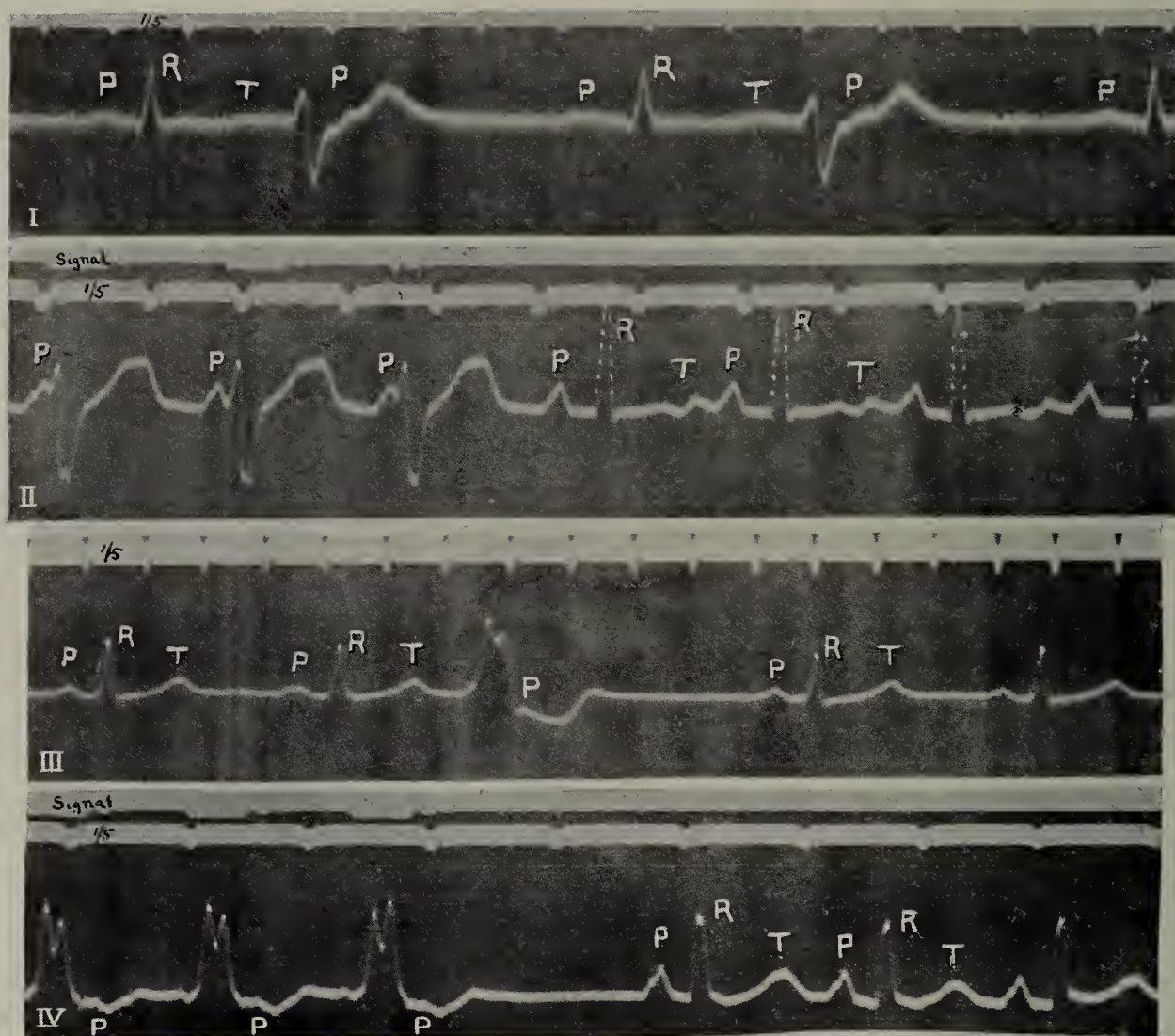


Fig. 117 ($\times \frac{5}{7.5}$). Four electrocardiograms comparing premature contractions observed clinically and experimentally.

- I. Electrocardiogram from a patient showing a "bigeminy" due to premature ventricular contractions. Alternate *P* summits fall with the anomalous complexes. For comparison with II.
- II. Electrocardiogram from a dog. The excitation signal is shown above. The figure shows the last three beats of a tachycardia started at a point on the surface of the left ventricle (the left margin of the heart, at its mid-point). Three of the abnormal beats are shown. There was no retrogression, the *P* waves of the auricular systoles are falling in regular succession throughout. At the termination of stimulation the normal sequence returns and is shown in the last four cycles of the curve. For comparison with I.
- III. A single premature ventricular contraction taken from a patient. The sequential auricular contraction falls with it. For comparison with IV.
- IV. The end of a tachycardia produced by stimulation of the right ventricle (at a point on the right margin of the heart about the junction of its upper fourth with its lower three-fourths) in a dog. The ventricular beats retrogressed to the auricle; the anomalous auricular contractions are shown as slight oscillations (marked *P* below the electrocardiogram). The last three cycles of the figure belong to the returning normal rhythm at the cessation of stimulation. For comparison with III.

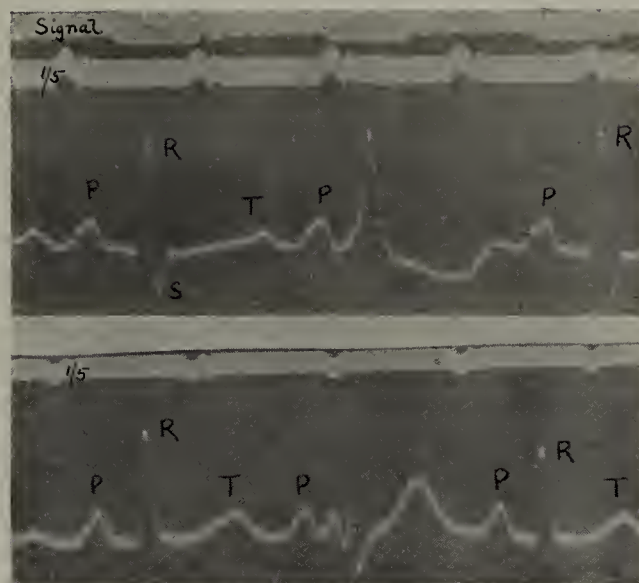


Fig. 118 ($\times \frac{2}{3}$). Two experimental electrocardiograms, each showing a single premature ventricular contraction, the first as a result of stimulation by an induction shock, the second spontaneous. The premature beats fall in each instance directly after *P*, giving rise to an *apparent* shortening of the *As-Vs* interval. The *P* summits are identified as such because they stand equidistantly from preceding and succeeding *P*'s. They are also recognised by measuring the ventricular complex of the normal beat and comparing it with the length of the anomalous complex. Taken from dogs. The signal in the upper curve shows three excitations; two are subminimal, the third is just sufficient to produce contraction. It is essential that threshold stimuli should be adopted or the curves suffer distortion as a result of the excitation current.

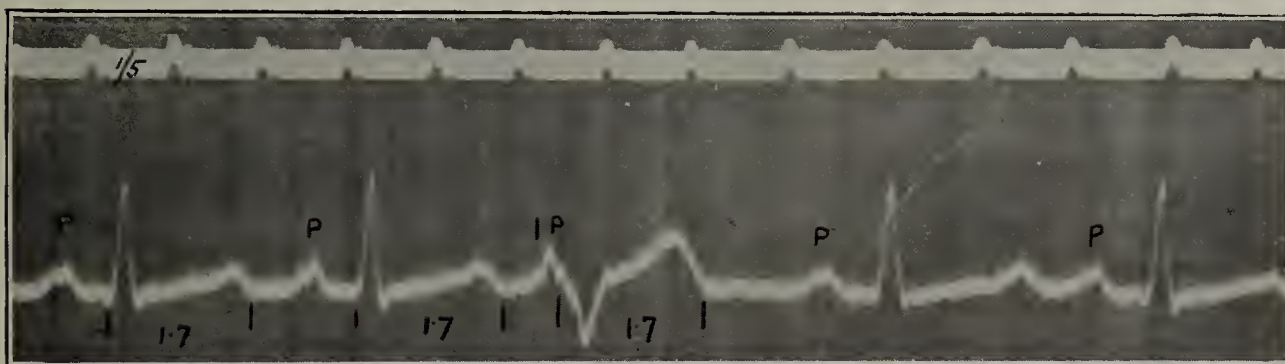


Fig. 119 ($\times \frac{5}{6}$). An electrocardiogram from a patient; showing a single premature ventricular contraction and a method of identifying an auricular complex which falls with it. The lengths of normal and anomalous ventricular complexes is the same; in this instance, 1.7 fifths. The portion of the anomalous complex which lies outside the limits of this interval belongs to auricular contraction.

point in diastole the contraction is excited. It means that the ventricular systole is always of approximately the same duration at whatever point its contraction is generated; and it may be of value in isolating auricular complexes which partially coincide with the ventricular (Fig. 118 and 119). Precisely similar measurements hold good in the instance of ventricular complexes following premature auricular contractions.

When excitations of the ventricle, applied to a single point of the musculature though at varying phases of diastole, are followed by ventricular complexes of several distinct types, the variation is due as a general rule to a curious phenomenon. The unexpected type is encountered when the excitation falls during the *As-Vs* interval, and it is the obvious result of interference produced by the normal impulses conveyed from the auricle (Fig. 120). Thus it may be said that in one and the same animal, excitation of a single point gives rise to curves of almost identical form at whatever point of diastole they may happen to originate,* provided that the contractions upon which they are dependent do not clash with other contractions, propagated through the normal channels.

Despite prolonged observation, the writer has failed to obtain ventricular complexes which are identical in outline when beats are propagated experimentally from corresponding ventricular points in distinct animals. The curves are unquestionably approximate duplicates. The work is difficult for it is quite essential that the heart should lie in precisely the same position relative to the electrodes in one animal and the next.

Anything but a rough localisation of the site of origin of premature ventricular contractions in man is impossible at the present time (Fig. 115 and 116), for we are only guided by results obtained in the dog in which the lie of the heart is different. It is probable that a closer localisation will be achieved in the future by a comparison of the curves yielded by the apes, and by the adoption of several leads.

The study is of present importance for it serves to identify beats which arise ectopically in the ventricle. When a ventricular contraction follows an auricular, a doubt may arise as to whether it is a response to it. But the electrocardiogram always differentiates the supraventricular from the ectopic ventricular contraction (Fig. 118 and 121).

It is also of service in another way. For it acquaints us with the fact that in most patients who exhibit premature contractions (and this applies to the auricular as well as the ventricular form) the beats spring almost constantly from a single focus. It is only in the exceptional case that they spring from several foci. It is also apparent that the focus of disturbance is remarkably constant from day to day, from month to month, and even year to year.⁵ These facts are recognised by the constancy of the shape and dimensions of the anomalous complexes as they are encountered in a single patient, at a single examination or at repeated examinations from time to time.

* Occasional exceptions are found.

The conclusion is the more important because it clearly indicated that the seat of disturbance from which heterogenetic beats are liberated is a very restricted one in an individual case.

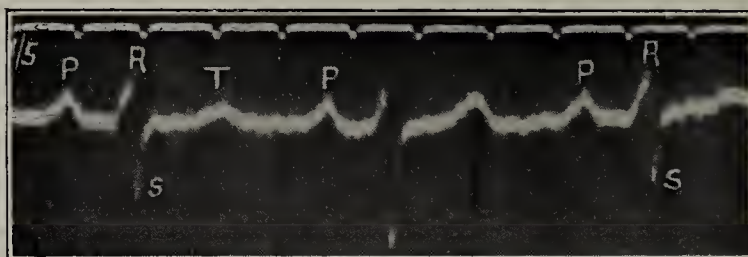


Fig. 121 ($\times \frac{2}{3}$). Electrocardiogram from a patient the subject of premature ventricular contractions. The figure illustrates the possibility of identifying premature ventricular contractions which occur very late in diastole. The central complex does not represent a response to the preceding auricle. The $As-Vs$ interval is very slightly reduced, and the ventricular complex is anomalous. Other premature beats from the same case gave similar electric complexes but many stood earlier in diastole. The shape of the premature complex is not widely different from those of the rhythmic beats, a consideration which leads to the belief that it has originated in the junctional tissues.

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CHAPTER XIV.

HETEROGENETIC RHYTHMS (*SUCCESSIVE PATHOLOGICAL CONTRACTIONS AND TACHYCARDIAS OF VENTRICULAR ORIGIN*).

PERIODIC acceleration of the heart's rate, a frequent clinical phenomenon, has its origin in a variety of causes. Sudden accessions of the rate at which the pace-maker elaborates impulses occur under purely physiological conditions, for example under the influence of emotion or during exercise. And in particular types of patients, more especially those who exhibit instability of the nervous system as a whole, a slight excitant may lead to tachycardia accompanied by sensations of distress; a marked and unexpected response of the heart, in the form of increased rate, to relatively slight disturbance is also found where there is reason to believe that the myocardium is unhealthy; for example, in the general toxæmia of tuberculosis and in other conditions.

But apart from accelerations of these and similar natures, a specific type of tachycardia exists in the human subject, which demands separate and careful consideration. Usually a grave malady, generally irremediable and not infrequently directly fatal, it calls for strict isolation when its pathology is studied. It is usually characterised by the severity of its symptomatology, the abrupt onset and offset of the attacks, and by the seemingly haphazard manner in which the crises are provoked or are assuaged. Treated from the pathological standpoint and as a tachycardia, it stands as a definite entity. Yet the mechanism of its production falls into line with many other disturbances of the cardiac rhythm. It is essentially a cardiac disorder, and consists of nothing more than the successive occurrence of those beats, which in preceding chapters have been designated pathological or heterogenetic.

The hypotheses, which assign the attacks to the withdrawal of tonic vagal influences¹¹ or to accessions of sympathetic central influences, are signally insufficient, a fact which will be clearly established subsequently.

Many years ago an ingenious theory was advanced by Hoffmann,⁶ who sought to prove that the tachycardia as it occurs in the patient is in reality representative of the usual cardiac rate of the subject investigated, and that the apparently normal periods between such attacks are due to a failure of transmission of alternate contractions from an upper to a lower heart chamber. His view was based upon an erroneous induction from scanty premises; he stated he found a simple mathematical ratio between the rates during fast and slow periods. Further observation has shown that such a relationship exists but rarely, and it has been proved experimentally that, where it exists, Hoffmann's hypothesis is untenable (Fig. 122). His view is now

only of historic interest. Yet it may be noted that we return to-day to an explanation previously expressed and subsequently discarded by the same author, namely the identity of the underlying factors in the case of single and successive premature beats.⁵

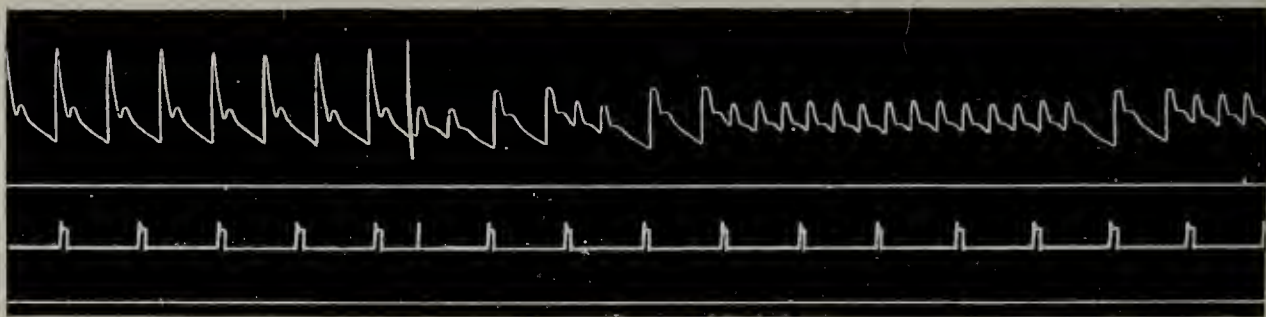


Fig. 122 (*Heart*, 1909-10, I, 65). A Hürthle carotid pressure curve from a cat in which the right coronary artery had been tied. To the left, a pulse curve is seen, which corresponds to the normal rhythm. To the right, the onset of a paroxysm of tachycardia is shown. The rate during the tachycardia happens to be exactly double the original rate of the normal rhythm. It is obvious that the rhythm to the left is not halved by any failure of impulse transmission. The time is in seconds.

The experimental production of heterogenetic rhythms.

Just as a single premature contraction is provoked by excitation of any portion of the cardiac musculature, so a regular series of contractions of like nature may be excited by the utilisation of suitably spaced induction shocks (Fig. 123) or mechanical stimuli. The production of tachycardia by means of spaced induction shocks is essentially artificial, though at the same time the experiments yield important information. The onset and the offset of such a tachycardia is abrupt, and the pause which terminates it is comparable to that which follows the single premature beat.* The experimental findings also teach us that eventually, wherever the exciting agent is applied, the artificial rhythm will dominate the whole heart rhythm, provided that its rate exceeds that of the pace-maker. The sinus rhythm becomes submerged, to reappear when stimulation ceases. And the new rhythm takes precedence to the old, whether auricle or ventricle is excited; in the instance of the latter the contractions are retrograde from ventricle to auricle.

When pathological beats arise from any cause in the ventricle, the transmission of the ventricular impulses in a direction the reverse of normal is accomplished with difficulty. A single beat, nay three or four such contractions, rarely produce a single auricular response; but when a longer

* The length of the pause is variable. If the auricular rhythm is undisturbed the ventricle awaits the first rhythmic auricular impulse, which will reach it after the cessation of tachycardia, and this impulse may fall at very varying intervals in relationship to the last beat of the tachycardia.

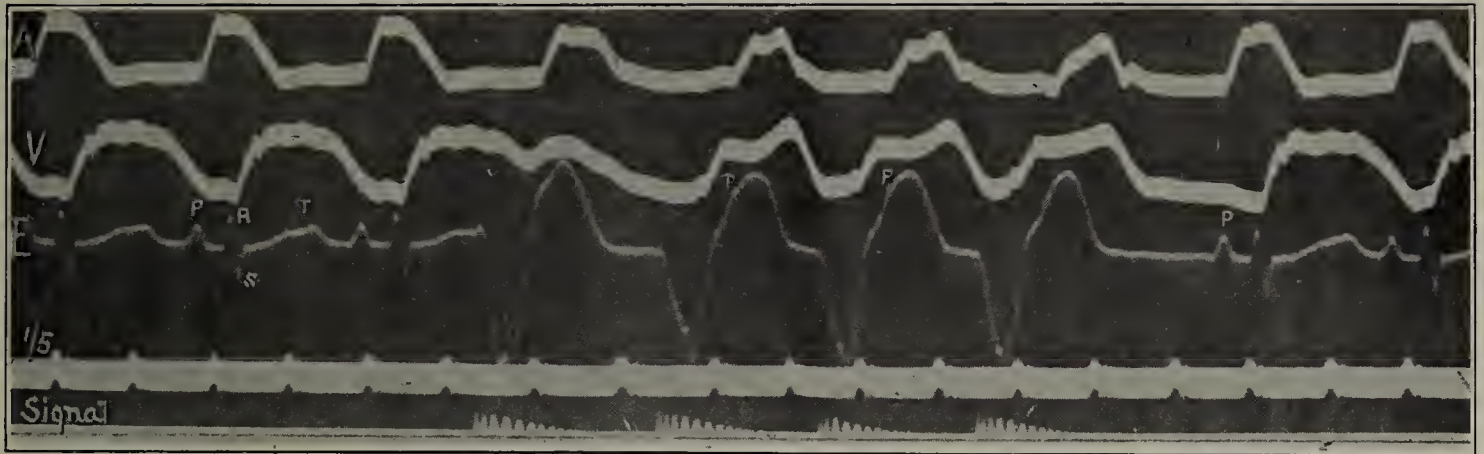


Fig. 123 ($\times \frac{2}{3}$). Simultaneous myocardigraphic curves (A =auricle, V =ventricle) and electrocardiographic curve from a dog. Showing a series of four rhythmic contractions excited (see signal) from the apex of the left ventricle (the actual point lay 1 cm. from the apex and on the left margin of the heart). There is no disturbance of auricular sequence, the P 's fall upon the anomalous ventricular complexes at the expected points (two of them are marked). The anomalous ventricular complexes consist of downward (apex-negative) and upward (base-negative) phases. From the same animal as Fig. 120.

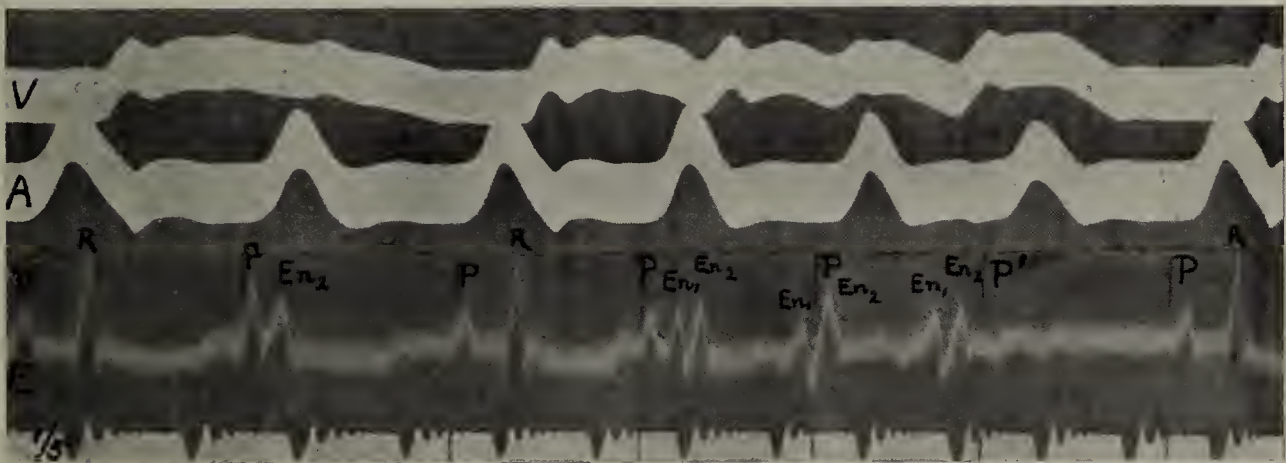


Fig. 124 (*Heart*, 1909-10, I, 122). Myocardigraphic curves (V =ventricle, A =auricle) and electrocardiogram from a dog in which the right coronary artery had been tied. To the left a beat of the normal rhythm is shown (P , R). The next systole of the auricle falls with a premature ventricular contraction (En). The next beat belongs to the normal rhythm. A series of three premature contractions follows, each consists of summits En_1 and En_2 ; P falls just before the first and upon the second anomalous complex. The third premature beat disturbs the auricular sequence (see myocardiogram). The electric complex which corresponds to the early auricular systole is anomalous (it is partially inverted). The curve ends with a normal heart cycle.

succession is provoked, sooner or later the auricular rhythm is disturbed; eventually it becomes submerged and the auricle finally responds to each ventricular impulse. The observations in regard to retrogression of the wave of heart contraction are uniform in the main. It is almost universally acknowledged that transmission is easier in the forward than backward direction^{1 & 10}; it is usually held that retrograde transmissions may be educated;⁹ it is known that all grades of reversed heart-block may occur; it is unquestionable that the circulation may be sustained, for intervals of minutes or even hours while such a mechanism is present.

Considerable interest centres around the auricular electric complexes of the retrograde beats. They fall with the ventricular complexes and are consequently difficult to disentangle in a pure form. Where they have fallen upon an almost horizontal effect (as in Fig. 124) they have had an isoelectric or inverted outline. Similar curves in which they are embedded in the ventricular complex are shown in Fig. 130; Hering⁴ has recently published curves of the same kind. We should have been led to expect a completely inverted type of complex, but apparently it does not occur; at all events, the records so far obtained show it to be at the most only partially inverted. It must be remembered that we have but very few pictures of the complex in "nodal" beats, and that while complete inversion has been present in these, it may not be universally the case.

Rhythms similar to those excited electrically, though of a less artificial nature, in the sense that they show a closer relationship to pathological conditions, have been evoked by the creation of sudden rises of arterial pressure,³ by the exhibition of poisons such as digitalis, aconitine,² muscarine¹³ etc., and by ligation of the coronary arteries.⁹ The methods of production are therefore in every way parallel to the known means of inducing the single premature beat.

A further and expected parallel is exhibited in that rhythms of the nature described can be shown to occur independently of alterations in sympathetic and vagal nerve influence.^{3 & 9} They arise as a result of changes which occur in the heart wall.*

The production of heterogenetic rhythms in the ventricle.

Much light is thrown upon the development of those conditions which culminate in paroxysms of tachycardia, by a careful study of the phenomena which follow in the wake of experimental obstruction of one or other coronary artery. The train of events usually proceeds in a definite manner. The preliminary disturbances of the cardiac mechanism consist in the

* In clinical work the co-relation between single and successive beats is often striking. In a given case, there is generally a single point of origin, and instances are often encountered in which any interference, which influences the occurrence of one, has a similar effect upon the other. As an example, the effect of posture upon single premature beats and tachycardias may be mentioned; it is often profound, one and the other may be present only when the patient stands. An increase of abdominal pressure (either in the standing or lying position) often temporarily abolishes both the interruptions of the slow periods and the paroxysms themselves.



Fig. 125 (*Heart*, 1909-10, 1, 104). Myocardiographic curves (V =ventricle, A =auricle) and Hürthle carotid pressure curve (C). A curve taken from a dog shortly after ligation of the right coronary artery, and showing premature ventricular contractions, single (e') and successive (e' , e''). In two instances a pair of premature contractions occurs, the second awakens an auricular response on each occasion; the premature auricular beats are marked with asterisks. An instance of the mechanism which precedes tachycardia of ventricular origin. Time in seconds.

appearance of single premature contractions (Fig. 92, 124 and 125), arising (in all instances of ligation of the descending branch of the left, and in most instances where the blood-flow is hindered in the right vessel) in the ventricle. As the nutrition of that portion of the heart wall, which is damaged, becomes more and more impaired, these heterogenetic contractions succeed each other at shorter and shorter intervals; soon they appear successively and rapidly in groups of two, three or more beats (Fig. 124 and 125). At a later stage, larger groups are found. When a number, perhaps two, three, five, ten or fifteen beats, follow each other, disturbances of the auricular rhythm are also noticed, as a result of retrogression. At a still later period all such beats are retrograde, that is to say, each is transmitted backwards in turn, and supplies the impulse which causes auricular contraction (Fig. 126). Fully established, a tachycardia of this kind (Fig. 127) may last from a few minutes to half an hour or more. The heart may fail to recover from it, but more frequently the old pace-maker is re-established, and its rhythm becomes dominant with the reduction of rate. A heart may continue to pass in this manner through many crises until the circulation is ultimately brought to a standstill, or the experiment is abandoned. Often the tachycardia itself progresses in rate; in the dog, the new rhythm, which is almost always conspicuous for its regularity, not infrequently surpasses 300, and may even reach a rate of 420 per minute (Fig. 128). Thus the elaboration of the contractions proceeds from the occasional to the frequent, and the successive beats when established, quicken in rate until the contractions of the ventricle follow each other so speedily, and the systoles diminish so markedly in amplitude, that the beats are ineffective in maintaining the circulation.* It is at this time that true fibrillation of the ventricle is usually established, a condition in which the musculature no longer beats co-ordinately, but in which it presents a tremulous and flickering activity. And it is often impossible, either by inspection or by the most delicate method of record, to ascertain the instant at which one condition, the rapid co-ordinate beat, is lost and is replaced by the other, the delirium which succeeds it.

The succession of events through which the ventricle passes is characteristic; there is an ever increasing state of irritability, a growing dominance of contractions of an obviously heterogenetic form. In the first place these phenomena are of importance in demonstrating the transition from a condition in which single impulses are generated to a state in which they arise in multiple fashion; the single beats and the successive beats spring, as may be shown electrically, from one and the same focus (Fig. 124 and 128); examined by every known method they appear to be of the same nature. Secondly, they are noteworthy because they exemplify the *spontaneous production of a regular and rapid rhythm of an evidently*

* When the ventricle is beating its fastest, it is not uncommon to find heart-block, partial or complete, established between it and the auricle (Fig. 127), so that the auricle drops beats, responds to 1 in 2, 1 in 4, or 1 in 6 (Fig. 128) ventricular contractions, or fails to respond to any.

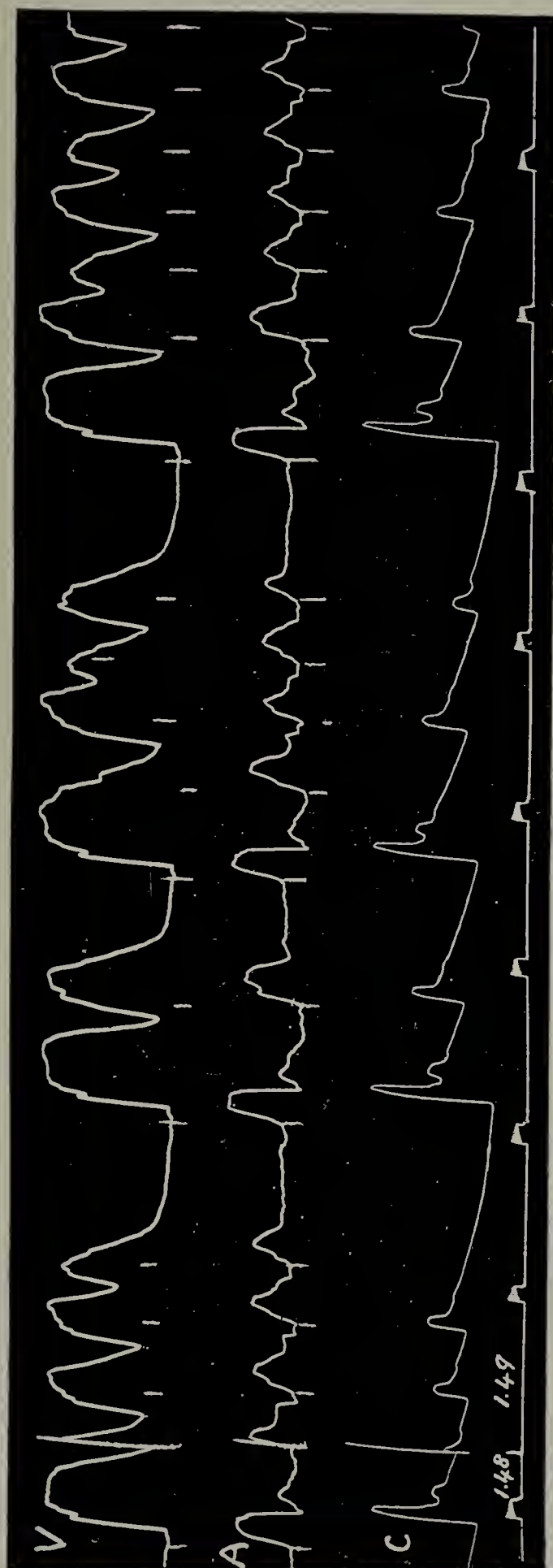


Fig. 126 (*Heart*, 1909-10, I, 105). Myocardiographic curves (V =ventricle, A =auricle) and Hürthle carotid pressure curve (C) from a dog in which the right coronary had been tied. The events are those which occurred directly before the onset of a long paroxysm of tachycardia, the commencement of which is seen in the last six beats of the figure. The earlier portions of the figure show runs of premature ventricular contractions (most of which regress to the auricle) with occasional breaking back to the normal rhythm at the end of the long pauses. Briefly, there are short preliminary paroxysms before the final long paroxysm is established. Time in seconds.



Fig. 127 (*Heart*, 1909-10, I, 112). Myocardiographic curves (V =ventricle, A =auricle) and Hürthle carotid pressure curve (C). To the left of the index marks one normal and one premature ventricular contraction are shown. To the right (one minute later) the ventricle is in tachycardia and the auricle is responding to each second or to two in three ventricular beats (reversed heart-block). The ventricular rate is approximately 220. The disturbances were the result of obstruction of the right coronary artery. From a dog; time in seconds.

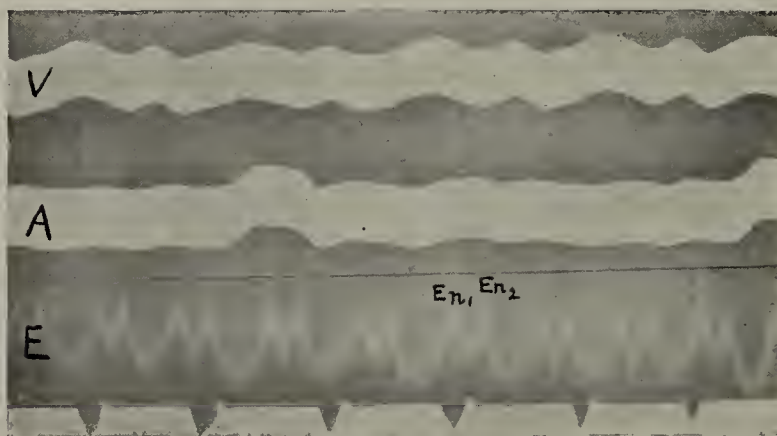


Fig. 128 (*Heart*, 1909-10, I, 124). Simultaneous myocardiograms (V =ventricle, A =auricle) and electrocardiogram from the same animal as Fig. 124. The ventricle is beating at a rate of 420 per minute. The auricle responds to every sixth beat. Each ventricular complex is of the same form, showing that the tachycardia originates in a localised area, and the form is the same as that of the premature contractions shown in Fig. 124,

ectopic character; for it can be shown beyond doubt in given instances that the impulses are formed in the ventricle. Thirdly, they require attention because the crises occur in paroxysmal fashion; contractions are propagated from two points; one the old pace-maker, the other lying in the ventricle; the former are homogenetic and the latter heterogenetic.

The paroxysms are also of importance because the pathological beats of which they consist are formed in the heart wall, and are evidently the result of local disturbance, and in no way attributable to altered central innervation.* Further, the mechanism of the attacks is definitely parallel to that presented by the clinical tachycardias, which we shall proceed to consider. The paroxysms commence abruptly in premature contractions, they cease with equal abruptness, and are followed by the characteristic pauses.

Heterogenetic rhythms arising in the ventricle of patients.

In dealing with a new and pathological rhythm which originates in the ventricle, the study is simplified by the remoteness of the seat of disturbance from the pace-maker. It is recognised as ectopic with facility, its rate proclaims it as pathological. But it is rare.

In patients who exhibit single premature contractions arising in the ventricle, occasional runs of two or even three or more successive beats of similar character are not infrequently recorded.^{8 & 12} On rarer occasions a case is encountered in which the succession is maintained for longer periods. Some examples of radial curves have already been given in illustration of the absence of auricular rhythm disturbance which may be found under these circumstances (Fig. 27). A series of curves from the same patient is shown in Fig. 131, and illustrates the points referred to.

In the electrocardiogram (Fig. 132) a short and regular paroxysm of six beats following each other at a rate of 210 per minute is seen interrupting the normal rhythm. The identification of the focus of origin of the anomalous contraction rests upon our experimental knowledge of the type of electric curve to which stimulation of various portions of the ventricular walls gives rise. In this instance we are dealing with contractions originating in the right or basal portion of the ventricle (Fig. 133). The figure allows a comparison of single and successive interruptions, which are of similar form and have consequently arisen in a single area. The six beats are also readily identified as ventricular by studying the time relationships of the single interruption succeeding them. Here the auricular contractions may be plainly seen. The recognition of the

* Section or non-section of the vagi is without effect upon their occurrence, or upon the temporary returns to the normal rhythm.

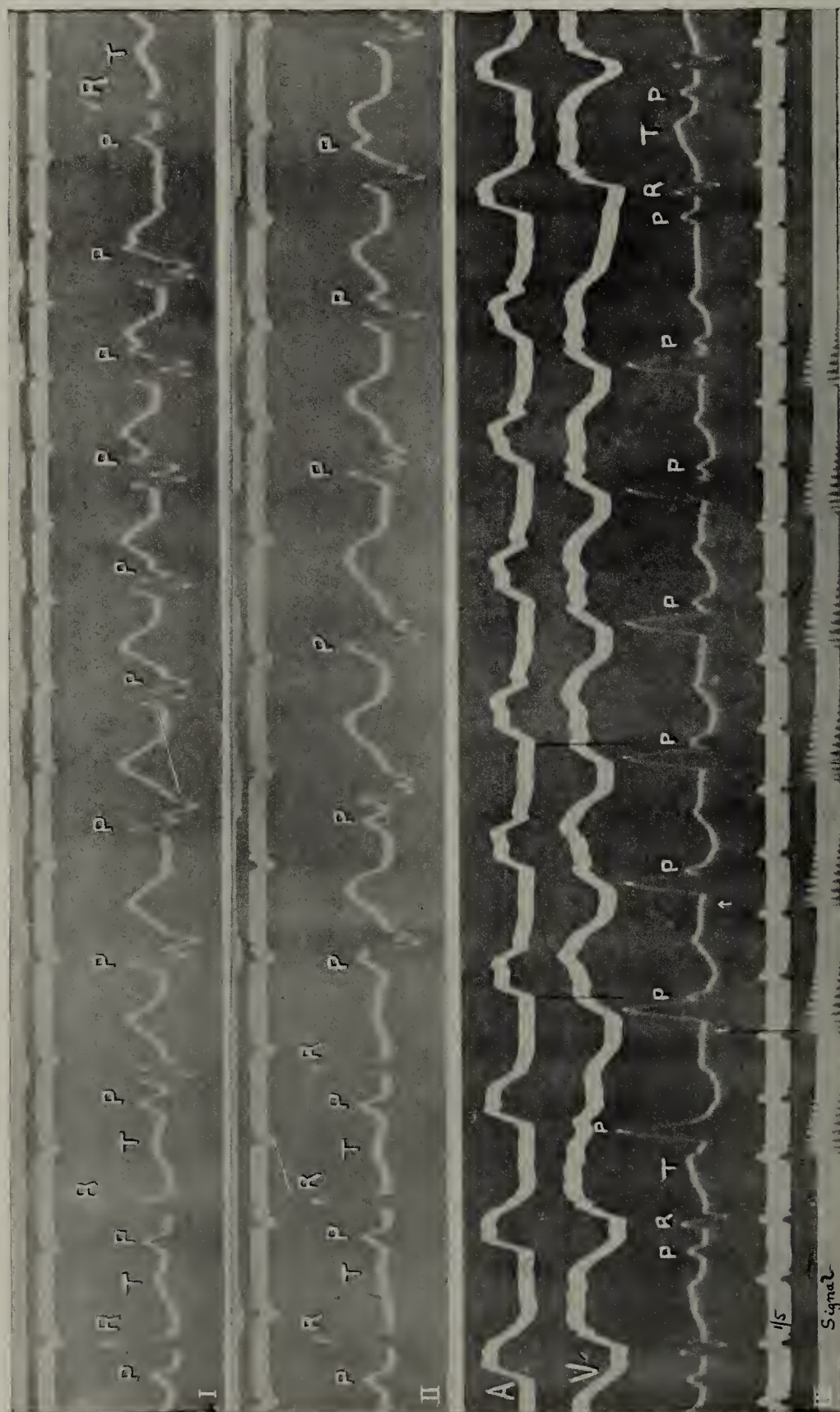


Fig. 129.

Fig. 129 ($\times 17$). Three experimental electrocardiographic curves from two dogs. In all such experiments, in which direct records were not taken simultaneously, the chest wall and pericardium were intact; the upper two curves are from the same animal. The lowest curve is from a different animal and simultaneous myocardiographic curves from auricle and ventricle are shown.

The first curve illustrates the type of beat obtained on internal stimulation of a large anterior papillary muscle in the right ventricle, (the signal of excitation is shown above). The normal cycles are followed by a regular sequence of abnormal ventricular complexes, upon which the regular *P* variations are superimposed; at the end of the curve a single cycle of the returning normal rhythm is seen. Stimulation of the same muscles in another animal yielded similar curves (Fig. 130 / *V*). The stimulation was effected through the superior vena cava.

The second curve is of a similar nature and from the same animal. The right ventricle was stimulated in the centre of its anterior surface (2 cm. from the intraventricular groove and 3 cm. from the pulmonary artery). While the first and second curves were taken, the chest wall and pericardium were closed and the heart lay in a natural position.

The third curve depicts a similar tachycardia excited from the right ventricle, (the mid-point of the right heart margin). The auricular rhythm is not disturbed, the *P* variations may be followed throughout, and their time relationships may be checked by comparing them with those of the auricular myocardiograph. The signal of excitation is placed below in this figure.

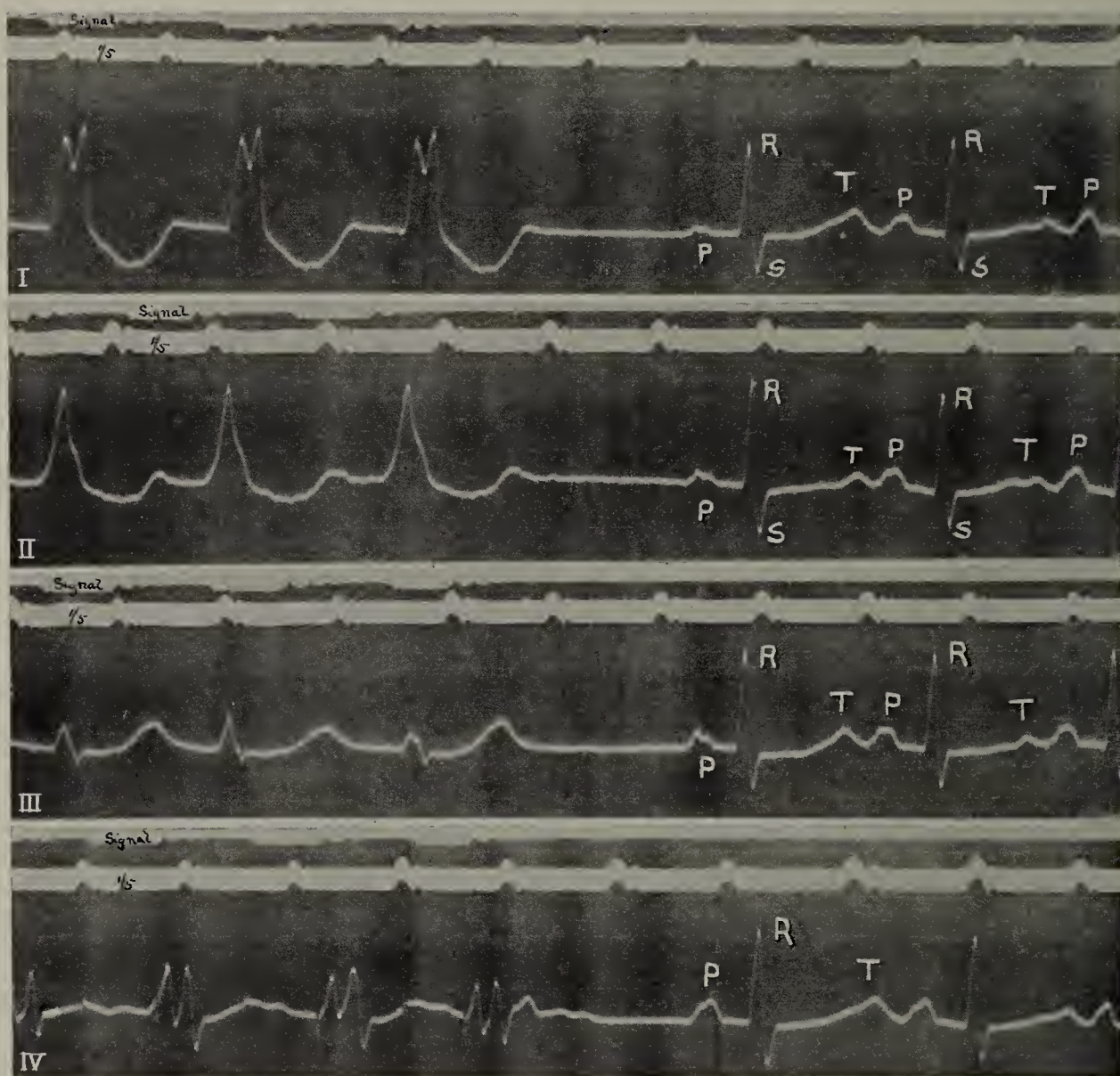


Fig. 130 ($\times \frac{5}{4}$). Four electrocardiographic curves from a dog in which pericardium and chest wall were closed. Each shows similar events. To the left in each strip are three beats of the termination of a paroxysm of tachycardia, excited by successive induction shocks from a single point of the ventricular musculature. In each case the tachycardia has been maintained sufficiently long to establish reversal of the rhythm, the auricular complexes are consequently indistinct during the tachycardias, for they fall with the anomalous ventricular complexes. To the right are two beats of the returning normal rhythm at the cessation of stimulation; (the signal of excitation is shown in each curve). The first three points of stimulation were in a line drawn from the mid-point of the base of the right ventricle in front, to the apex.

- I. Stimulation of point mid-way between this base and the intraventricular groove.
- II. Stimulation of the groove itself.
- III. Stimulation of a point mid-way between the groove and the apex of the heart.
- IV. Stimulation of large anterior papillary muscle inside the right ventricle.

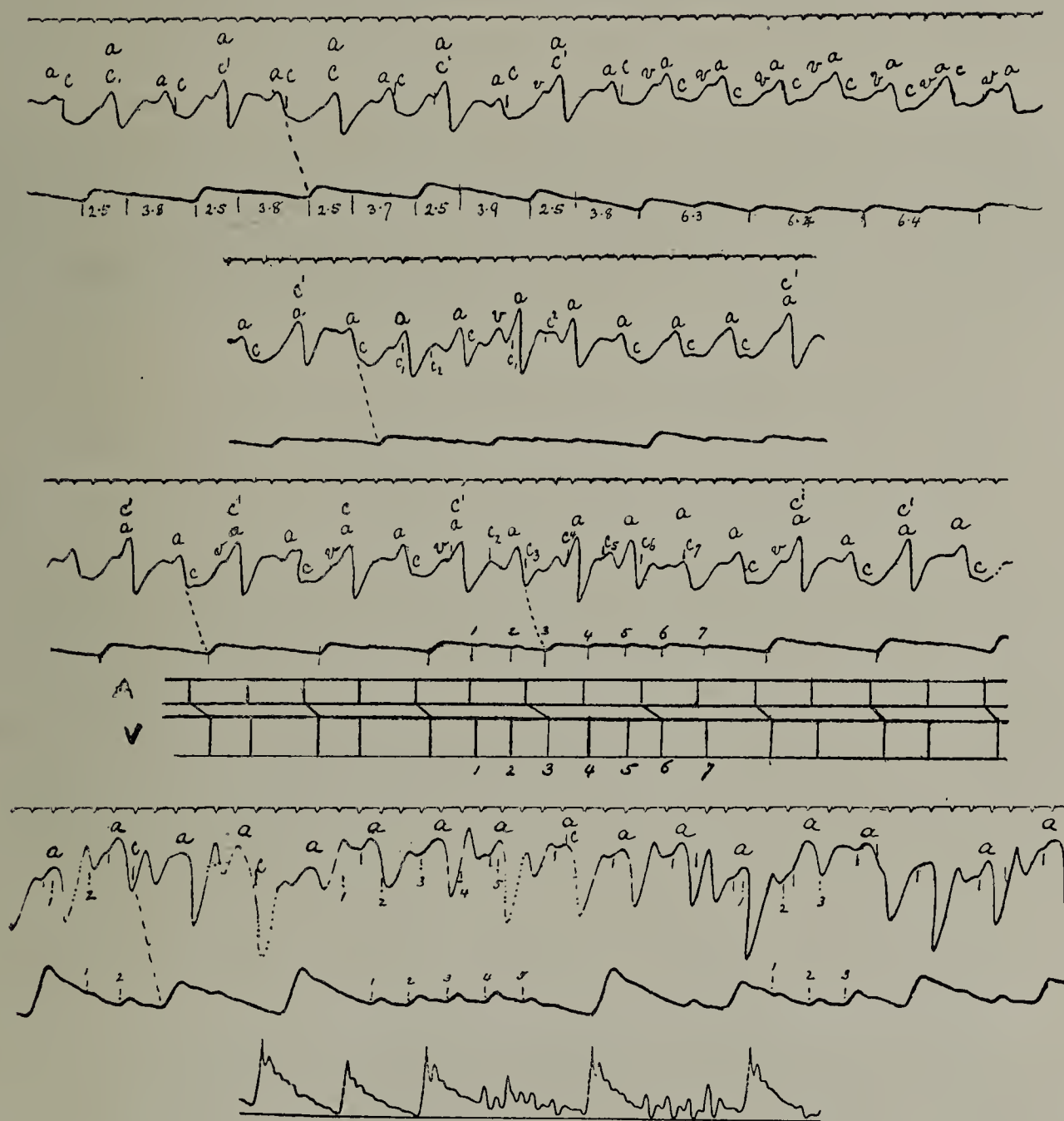


Fig. 131 ($\times \frac{1}{5}$) (*Lancet*, 1909, I, 382). Four polygraphic curves and one radial curve from a case of multiple premature ventricular contractions. The first strip shows a bigeminy passing into the normal rhythm. The second strip shows a pair of premature beats. The third strip a bigeminy passing into a more complex condition of which the analysis is uncertain, but in which the whole disturbance results from premature ventricular contractions. The fourth and fifth strips show still more complex irregularities, each the result of a similar mechanism, namely premature ventricular contractions. Electrocardiograms from this case are illustrated by Fig. 115. Radial curves are also shown in Fig. 27.

auricular contractions during the fleeting tachycardia is difficult after the first beat, but measurement makes it obvious that retrogression has not occurred, for the first auricular beat of the returning normal rhythm falls at the expected point.

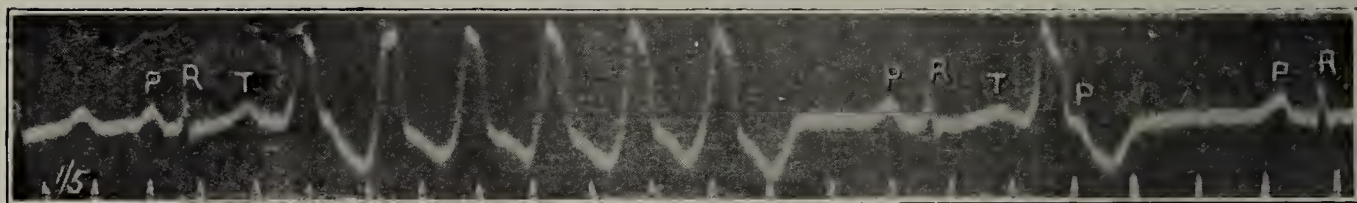


Fig. 132 ($\times \frac{2}{3}$). An electrocardiogram from a patient. The normal rhythm is interrupted by a paroxysm of six premature ventricular contractions and a single premature contraction of the same type. (Fig. 58 is from the same case.)

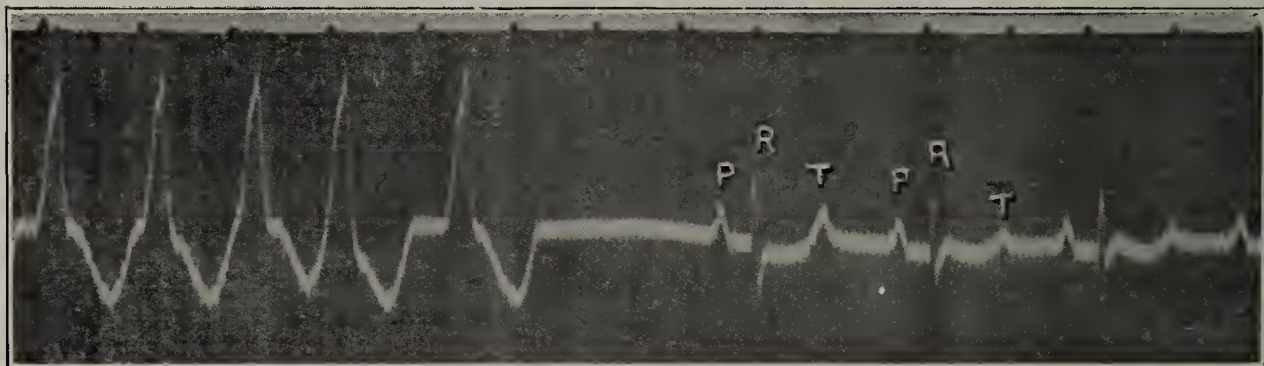
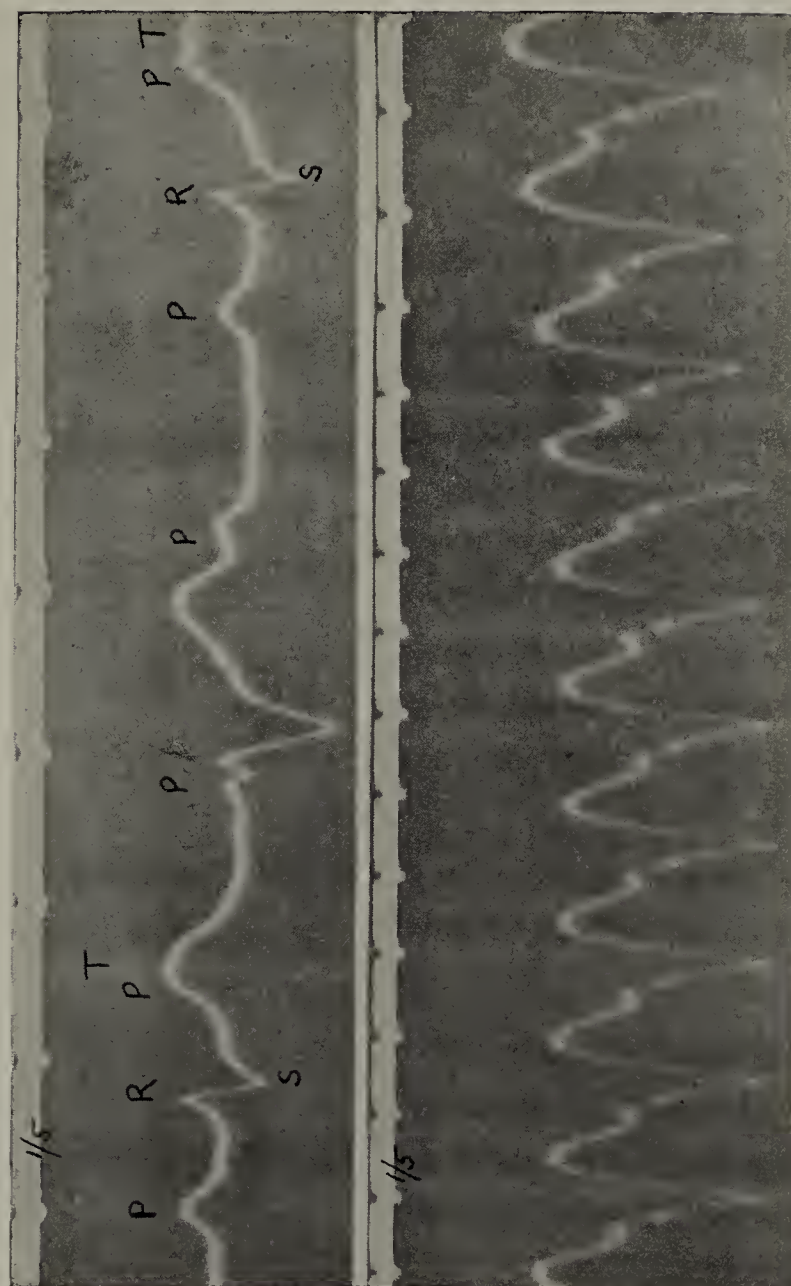
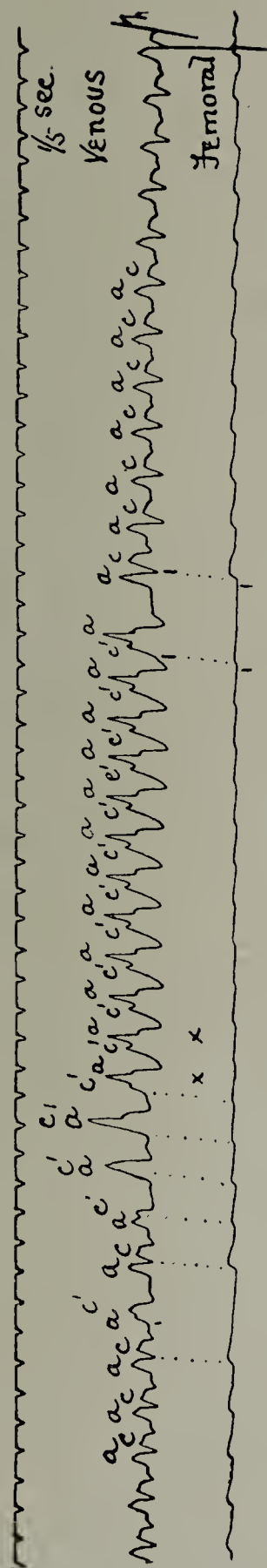


Fig. 133 ($\times \frac{5}{6.5}$). The end of a paroxysm excited by weak faradisation of the front of the right ventricle of a dog's heart. For comparison with the last figure. The normal beats, returning at the cessation of stimulation, are seen to the right. The regular and rapid oscillations in this figure were the result of visible muscular tremor.

Fig. 134 (*Heart*, 1910-11, II, 141). A polygraphic curve, from a dog, showing the form of venous and femoral curves during a tachycardia of ventricular origin (excited by successive induction shocks). To the left the normal rhythm is seen, each beat is accompanied by *a* and *c* waves (*a* and *v* are compounded). The rhythm is first interrupted by a single premature beat *c'*, one normal cycle follows, and then the ventricular tachycardia commences. It becomes retrograde at the fifth beat and from this point onwards the venous pulse cycles are uniform in appearance, and the "ventricular form of venous pulse" is present.

Fig. 135 ($\times \frac{5}{7}$). Two electrocardiographic curves taken within a few minutes of each other, from a patient the subject of transient attacks of tachycardia. The upper curve shows a condition of 2:1 heart-block, interrupted by a simple premature ventricular contraction. The auricular systoles (*P*) to which there are no responses are superimposed upon the electric complex of the premature beat. The lower curve was taken at a slower rate, and shows a succession of beats, of a nature similar to that of the single premature beat of the upper curve. The paroxysm, which was at a rate of 200 beats per minute, lasted approximately five minutes.



An example of a longer paroxysm, probably originating in the apical and left portion of the ventricle, is exhibited in Fig. 135. The actual onset of the paroxysm was not observed: but its duration was approximately five minutes. It is the only example of a prolonged ventricular paroxysm which has come under the notice of the writer. The identification of the auricular contractions is impossible, but it seems probable that auricle and ventricle are contracting at the same rate, for each cycle is identical with those standing adjacent to it; whereas, if auricle and ventricle were contracting at separate rates the auricular complexes would superimpose themselves at haphazard intervals and destroy the symmetry of the curve.*

As in the experimental instance, so in man, each series of premature ventricular contractions tends to be followed by a pause of considerable length. Where the series is composed of but few beats, it may happen that they are in measure interpolated (Fig. 132), but where retrogression has occurred, a pause comparable to that following a premature contraction arising in the auricle is present. The pause marks the return from the new rhythm to the old.

The experimental tachycardias arising in the ventricle, and the isolated instances in man, are particularly serviceable in aiding our understanding of similar and more frequent conditions awakened in the auricle. Many parallels may be traced between the behaviour of one or other chamber in this respect, and when the full facts are considered, the evidence enforces the inclusion of auricular and ventricular tachycardias in the same or a similar category.

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* In this particular instance the chief argument against the presence of retrograde beats lies in the fact that heart-block is shown by the curves of the slow periods.

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- ¹³ ROTHBERGER (J.) and WINTERBERG (H.). "Über scheinbare Vaguslähmung (bei Muskarin, Physostigmin " etc.). *Archiv f. d. ges. Physiol.*, 1910, CXXXII, 233-254.

CHAPTER XV.

HETEROGENETIC RHYTHMS (*continued*). (*S U C C E S S I V E*
P A T H O L O G I C A L C O N T R A C T I O N S A N D
T A C H Y C A R D I A S O F A U R I C U L A R O R I G I N .)

Clinical tachycardias of auricular origin.

THOSE instances of grave disturbance of the circulation as a consequence of the periodic appearance of sudden accession of heart rate, accessions in which the rhythm is regular, are almost always the outcome of heterogenetic beats arising in the auricle. The proof of this proposition rests upon an examination of numbers of individual cases. Our knowledge of tachycardias of auricular origin is consequently more complete than it is in regard to those of the ventricular type. The rarer ventricular paroxysms have been utilised in the preceding chapter that the comparison with the experimental condition might be closer.*

In patients who exhibit regular and paroxysmal tachycardia, intervals of slow and fast heart rate of varying duration, succeed each other. The periods of tachycardia are usually less prolonged than the periods of comparatively slow rate, but no general statements can be made as to the relative duration of one or other, or as to the actual length of the paroxysms themselves. The periods of tachycardia last in one and the same patient, or from one patient to the next, from a few seconds (Fig. 136 and 137) to an hour, several days or even weeks or months; the main features of the paroxysms are identical, whether they consist of short successions of three, five or ten beats, or whether they are maintained for long stretches of time. Apart from the duration, the long and short paroxysms differ only in so far as the long crises tend to produce general and increasing circulatory embarrassment, and in so far as the continued attack may be accompanied by signs of functional failure of the heart.

The recognition of tachycardia arising in the auricle is accomplished with facility by means of polygraphic tracings (Fig. 137). The venous curves show distinct and often prominent *a* waves preceding each *c* wave. The auricular systole frequently coincides with the preceding ventricular beat, and exaggerated waves, to which attention has already been drawn, result; (Fig. 137*b*, 137*c* and 138).

* The writer bases his assertions upon a personal and instrumental examination of 20 cases, in all of which prolonged paroxysms occurred, and upon the curves published in the papers of the accompanying bibliography.

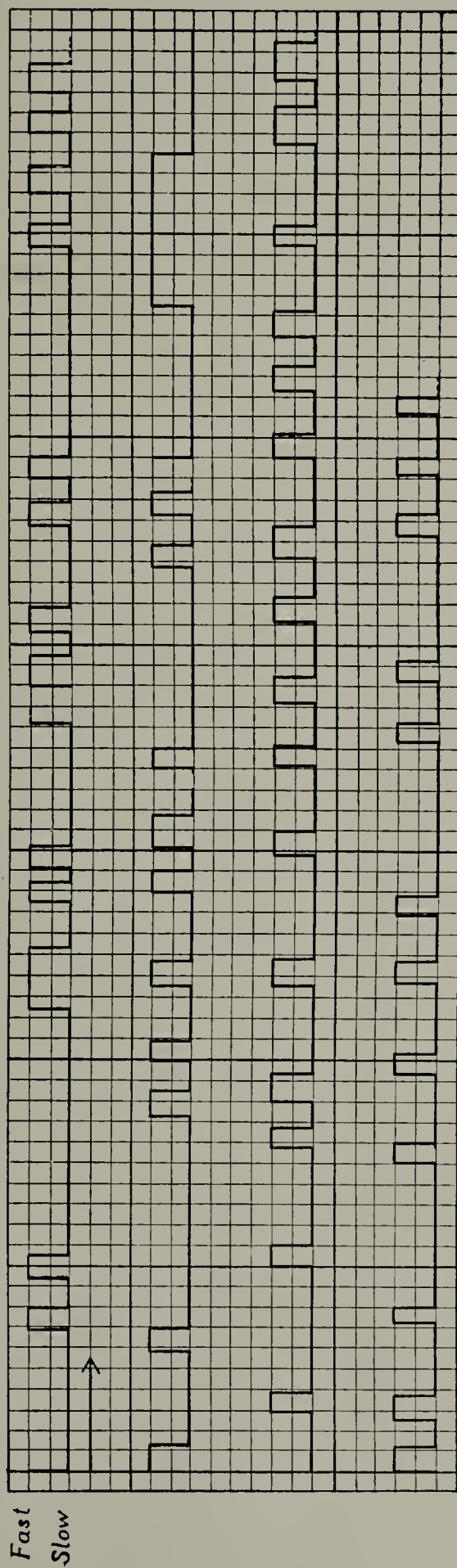


Fig. 136 (*Heart*, 1909-10, I, 263). The chart shows, in diagrammatic fashion, the relative duration of tachycardial and slow pulse rate in a patient the subject of short attacks. It has been constructed from a single curve, and covers 1 hour and 49 minutes. During this time 54 paroxysms occurred. The distance between adjacent ordinates is equivalent to 5 sec.. The diagram reads continuously from left to right and from above downwards in 4 lines, and the record is unbroken, except where short stops or obscure tracing are indicated by broken lines.

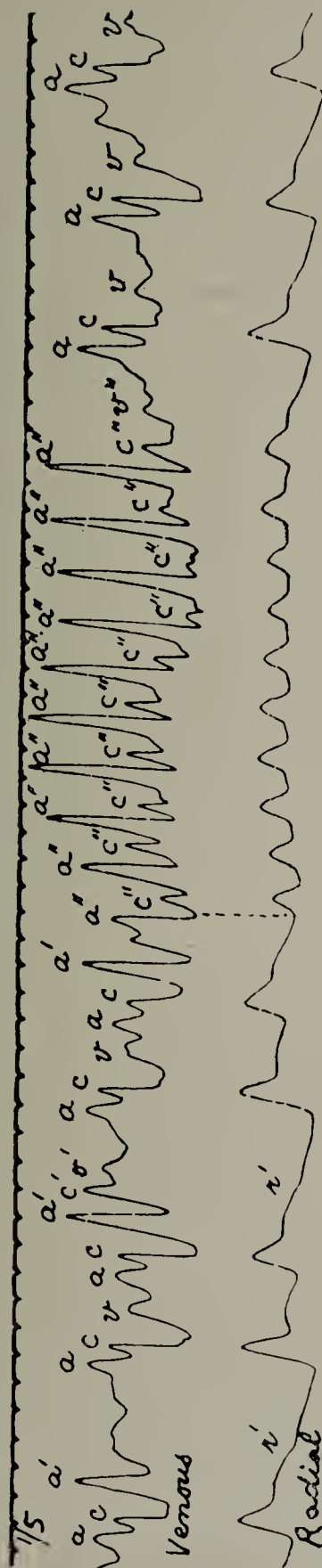


Fig. 137c (*Heart*, 1909-10, I, 265). Radial curve from a case of paroxysmal tachycardia. Showing three short paroxysms, the onsets and offsets of which were signalled by the patient. The slow periods are irregular. The pathological beats of slow (interrupting beats) and fast periods were of a similar nature. They all arose at a single point in the auricular substance, removed from the pace-maker. (Fig. 113, 136 and Pl. I, Fig. 215, are from this case.)

1/5-sec



Fig. 137d (*ibid*). Polygraphic curve taken at a slower rate and from the same patient. Showing that the interruptions of the slow periods are premature auricular contractions, and that the paroxysms are of similar nature.

In all these cases the paroxysms end in the characteristic post-paroxysmal pauses. Where the onset is shown, it is abrupt; there is neither a gradual transition from fast to slow nor slow to fast rate, neither is there a simple mathematical ratio between fast and slow rates.

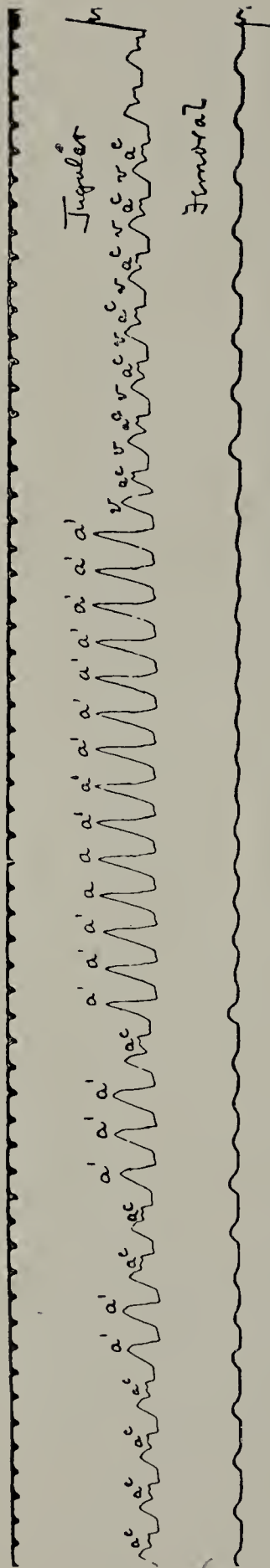


Fig. 138. Venous and radial curves from a dog; to show the forms of these curves during a tachycardia of auricular origin. The auricle was stimulated by means of successive induction shocks. The normal rhythm is disturbed by a group of two and a group of three successive premature auricular contractions. Finally it is disturbed by a longer paroxysm of the same nature. The return to the normal sequence is seen at the end of the figure. Notice the height of the a' waves during the paroxysm. They are due to auricular systoles falling back upon the preceding ventricular systoles; coincidence of a' and v .

The transition of events, from the occurrence of a single premature contraction, to a run of a few successive beats and ultimately to established tachycardia, may be traced in a single patient*; as in the spontaneous experimental tachycardia a prolonged attack may be foreshadowed, or may be succeeded, by shorter paroxysms, which are similar to it (Fig. 137*b*). It is also observed that where the conditions predisposing to paroxysms are present, the slow rhythm is customarily interrupted by occasional or frequent premature contractions (Fig. 137, 139 and 140). This is generally the case in both the experimental and in the clinical types. Furthermore it is generally found that the solitary interruptions emanate from the same heart chamber as the individual beats of the paroxysms themselves. But to these rules exceptions are not uncommon, for it may be found that in a clinical case of tachycardia arising in the auricle, the slow periods are regular, or that on occasion they are disturbed by premature beats of ventricular origin; just as in the experimental tachycardia the slow periods may be interrupted by premature beats of auricular origin, while the tachycardia is ventricular. It may be said that the great majority of the beats which interrupt the slow periods and the individual paroxysmal contractions have a common starting point; and if it can be shown that the focus is ectopic, that is to say if it lies at a point removed from the pace-maker, the proof of which is generally forthcoming, we shall have strong presumptive evidence that the beats are all of the same nature,

* Clinical and experimental transitions have been noted in the instances of premature beats arising in the ventricle.

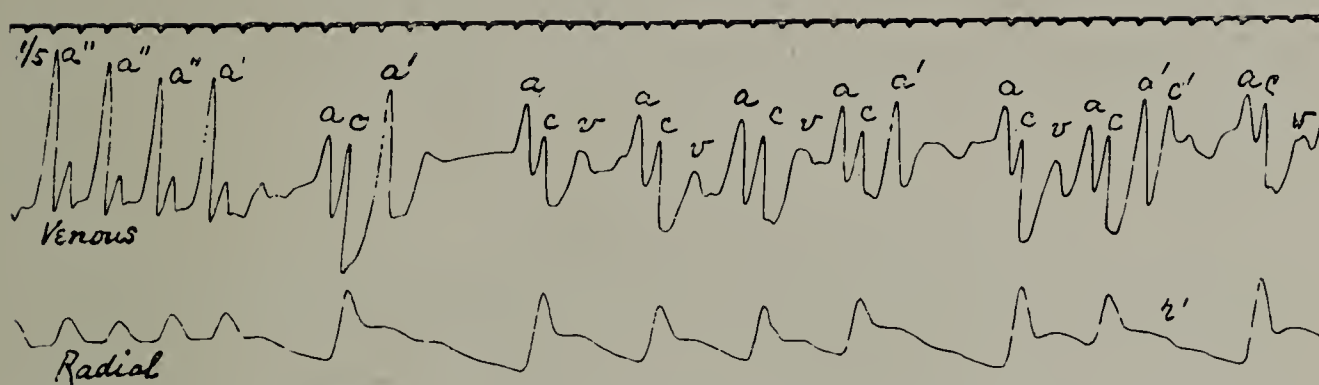


Fig. 139 (*Heart*, 1909-10, I, 267). Polygraphic curve showing the termination of a paroxysm of tachycardia of auricular origin. The paroxysm ends in a characteristic pause and this is succeeded by heart beats which gradually accelerate. The slow rhythm is interrupted by premature auricular contractions. From the same case as Fig. 137c etc..

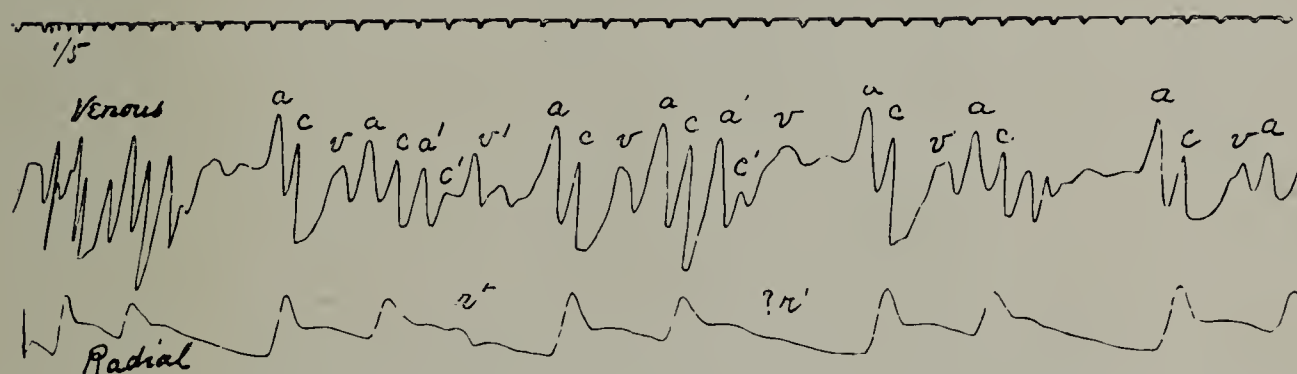


Fig. 140 (*Heart*, 1909-10, I, 267). Polygraphic curve showing premature auricular contractions (a'). From the same case as Fig. 137c etc..

i.e. that the paroxysmal beats like the single interruptions are generated heterogenetically.

The electric curves give us the evidence which we require. For not only can it be shown that the paroxysms are ectopic, but that the interruptions of the slow periods are also ectopic and arise at the same point; and thereby we are led to the conclusion that there is no essential difference between single interruptions and beats which form integers in a paroxysm. In brief, in the sense that the single premature contraction is heterogenetic, so are the paroxysmal beats. And this conclusion is established from a consideration of the rate of the new rhythm, and the character of its onset and offset. Each is part and parcel of the same phenomenon, namely the propagation of heterogenetic contractions from the auricle. In a preceding chapter the rarity of premature contractions arising at the pace-maker itself has been alluded to, and it has been attributed to the relatively small extent of this area, in comparison with the remaining heart musculature. It happens that in cases of auricular tachycardia which have been examined electrically, no instance of a tachycardia has

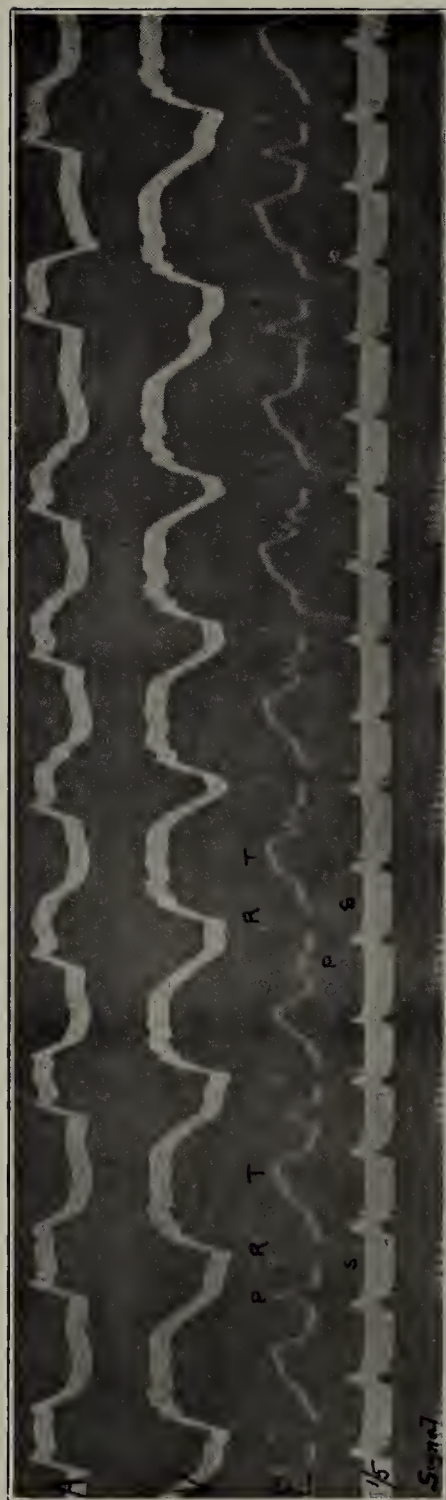


Fig. 141 ($\times \frac{2}{3}$). Simultaneous myocardiographic (A =auricle and V =ventricle) and electrocardiographic curves from a dog. Showing an auricular tachycardia excited (see signal) from the base of the appendix of the right auricle (1.5 cm. from the sulcus). The first three excitations fall during the refractory periods of the auricle, the next four produce responses, of which the auricular complexes are alone anomalous; they consist of bifurcated peaks. The return to the normal rhythm is shown at the end of the figure, where excitation ceases. From the same animal as Fig. 108.

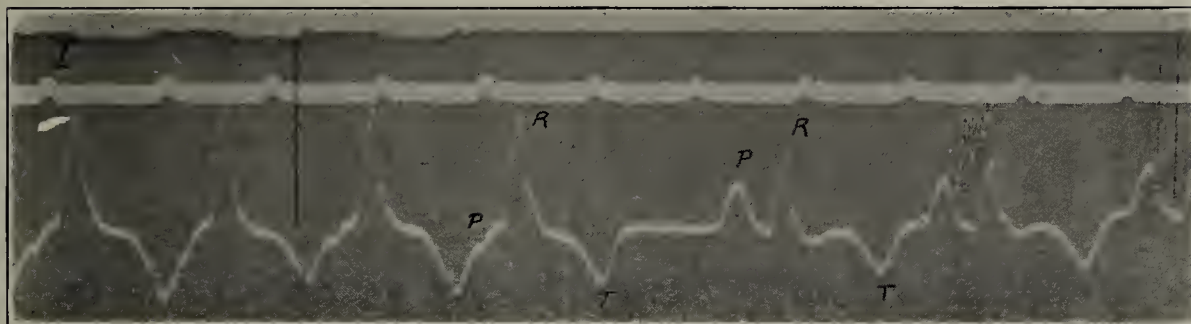


Fig. 142 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 23, *Fig. 7*). * An electrocardiogram from a dog showing the termination of a period of tachycardia, induced by stimulation of the neighbourhood of the inlet of the pulmonary veins (the signal of the induction shocks is shown). There are four paroxysmal beats and two normal beats in the figure. The *P* summits of the paroxysm are anomalous. The ventricular complexes show an increase of *R* and deepening of *T*. For comparison with the succeeding figure.

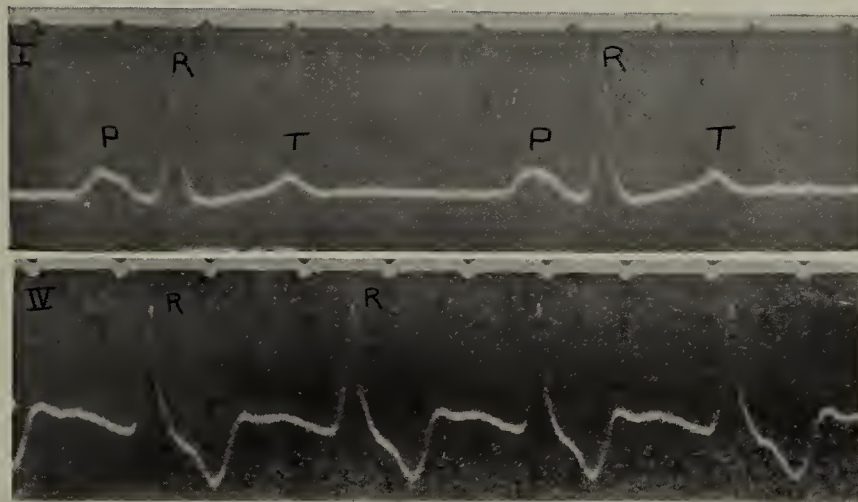


Fig. 143 ($\times \frac{1}{2}$) (*Heart*, 1910-11, II, 127, *Fig. 9*). Two electrocardiograms from a case of mitral stenosis and paroxysmal tachycardia. The first curve is from the normal and slow period. The second from a paroxysmal period. The paroxysms were of supraventricular origin. The auricular representatives are obscure. The anomalous ventricular complexes may be compared with those of the preceding figure. (Fig. 137*b* is from the same caso.)

been discovered where the seat of impulse formation could be shown to lie at the pace-maker; coupled with the knowledge that tachycardia is far less frequent than the single premature beat, the fact is not surprising. It is worthy of consideration, for although the ectopic quality of the observed tachycardias is of service in that it helps us to arrive in these individual instances at the conclusion that they are likewise heterogenetic, there is no *a priori* reason for believing that a similar heterogenetic production of rhythm may not have its seat in the pace-maker itself. The arguments are exactly parallel whether applied to single or successive beats.

The location of the point of origin of beats arising ectopically in the auricle has been fully discussed in Chapters IV and XIII. The accurate fixation of such points, much as it might be desired, is at present beyond our scope, but we have it in our power to show that a given auricular contraction arises at the pace-maker or otherwise, by studying the electric complex which represents the course pursued by the contractions in the auricle.

Paroxysms of tachycardia which are of supraventricular origin are not invariably recognised with facility, for the reason that an aberrant type of ventricular complex may be present as an accompaniment of the paroxysmal cycles. We have already noticed the variations in discussing single premature contractions which arise in the auricle. Fig. 142 is an exceptional and experimental example of the same phenomenon where the cycles are successive. The curve to the left is a record of a tachycardia propagated by induction shocks from the pulmonary veins. The ventricular complexes may be compared with those following the return of the normal rhythm (shown to the right, *R* is increased in the upward, *T* in the downward direction). The next figure illustrates similar events in a patient who suffered from paroxysmal tachycardia. *R* is increased and *T* is inverted during the paroxysm. For the full analysis of this case, the reader is referred to the original report of it.⁸ Where there is a doubt as to the supraventricular origin of the paroxysm, as in this case, a final conclusion may be arrived at by a careful comparison of other leads taken during slow and paroxysmal periods.

What has been set forth in the preceding paragraphs may be evidenced by a brief description of two characteristic examples of the affection.

Example of transitory tachycardia of auricular origin.

A radial curve, taken from an instructive instance of paroxysmal tachycardia⁶ arising in the auricle, is presented in Fig. 137*d*. The short runs of quick beats, periodically effacing the slow sinus rhythm, are very striking. Now this curve was taken from a patient in whom shorter or longer paroxysms were almost always present on examination. Their nature is clearly defined by the venous curves (Fig. 137*c*). The paroxysms are known to be auricular on account of the position of the tall *a* waves (compare the experimental curve Fig. 138); they are suspected of being ectopic for the pauses which succeed them are equivalent to those following premature contractions in the same patient; they are known to be hetero-

genetic for a similar reason, because the rate of the new rhythm is excessive and because it starts abruptly in a premature auricular contraction.

Electric curves from this patient illustrate this book at several places (Fig. 49, 113, 114, and 175). They afford us similar and additional information. The premature beats which disturb the slow periods are all of auricular origin, and the complexes which represent the contractions of the upper chamber are anomalous, consisting of short summits directed downwards. The premature contractions consequently arise at a point removed from the pace-maker. They are all generated in the same focus, for the auricular complexes are of constant outline. It will be noticed that the ventricular complexes accompanying the premature beats in this patient are of unusual types (Fig. 113), but this fact only concerns us at present in that it aids the interpretation of the paroxysmal curves. The paroxysm shown in Plate I, Fig. 215 opens with ventricular complexes of the type shown in Fig. 113. It is continued with complexes of a more normal type (also seen in a premature beat, the central one of Fig. 114). But the point of importance is that the auricular complexes are of a single type, and not only demonstrate that the paroxysm is unifocal in origin, but that the same focus supplies the impulses for single and successive premature beats. The auricular complex is always found superimposed upon the preceding *T* summit, namely during ventricular systole, a fact which we are led to anticipate from the height of the *a* waves in the venous curves. The auricular summits (*P*) are seen as isolated peaks in Fig. 175, where they fall clear, in alternate fashion, for here only each second contraction of the auricle awakens a ventricular response.

Example of tachycardia arising in the vicinity of the auriculo-ventricular node; nodal rhythm.

A second case is of equal importance in helping to establish our main contentions. The general type of mechanism is shown in the polygraphic tracings (Plate II, Fig. 216 and 217) one including a venous, the other an apex curve. The rate of the interrupting beats, the onset with a premature beat and the offset in a long post-paroxysmal pause are held in common with the first case described. But the point of origin is different. The *a-c* intervals of the venous curve are reduced from .2 sec., during the slow periods, to .06 during the paroxysms. The mechanism is one in which auricular and ventricular systoles of the same cycle fall together. The contractions of upper and lower chambers are therefore attributed to impulse formation in a central area, *i.e.* in the vicinity of the auriculo-ventricular node. The single premature beats of the slow periods were generally of a similar nature.

The interpretation of the curves is completely corroborated by the corresponding electrocardiograms. Plate II, Fig. 218 includes a whole paroxysm and at its termination shows premature beats interrupting the

restored slow rhythm. As in the previous case, the ventricular complexes of the paroxysms correspond to beats of supraventricular origin, for they are of the same general form as are those of the slow periods. But the presystolic event is peculiar in two respects: *P* is completely inverted during the paroxysm, and the interval *P-R* is markedly shortened (from .14 to .08 sec.). From the shape of the auricular complex we know that the impulse has arisen in the lowest levels of the auricular tissue (Chapter IV), an observation which is in harmony with the shortened *As-Vs* interval. The condition is closely approached by the experimental curves shown in Fig. 15 II.

The same mechanism is exhibited by the experimental curve (Fig. 144). We need not concern ourselves with the first half of this figure; it will suffice if attention is directed to the ventricular summits marked *R*,¹³ *R*¹⁴ and *R*.¹⁶ Before the first two, *P* is inverted, in the last upright. The shortening in the *P-R* interval is from .088 to .065 sec.. The value of the electrocardiogram is enhanced, for side by side with it are myocardiograms from auricle (*A*) and ventricle (*V*) and careful measurement allows an accurate comparison of the intervals and proves the simultaneous contraction of auricle and ventricle. Slight discrepancies in the degree of shortening in the simultaneous curves will be noticed, as will also the similar discrepancies of the shortening of *a-c* and *P-R* intervals of the clinical case; they are of significance, but it is impossible at the present time to enter into a full exposition of them; it has been attempted in the original report of this unique case.⁷ The evidence that the impulses are arising in the auriculo-ventricular node or its immediate vicinity may be briefly summarised.

1. The marked shortening of the *a-c* interval and the *P-R* interval.
2. The synchronous contraction of auricle and ventricle and the comparison with the experimental curves.
3. The complete inversion of the auricular complex.

The actual degree of shortening might be held as of importance, but it appears from the observations of Hering that the chief time loss in normal conduction is in the node itself and his measurements favour the view that a shortening of various degrees is not only compatible with, but may be characteristic of, the nodal origin of heart beats; and it seems to be true that so long as there is shortening the impulse arises in this situation, whatever the degree of shortening (Chapter XII). Simultaneous contraction of auricle and ventricle, which has been demonstrated at the end of vagal stimulation, and after destruction of the sino-auricular node,³ results in similar electrical effects.

The premature beats of the slow periods in the patient described were for the most part of a similar electric nature. An instance of one is shown in the third beat following the paroxysm. But beats of an aberrant type

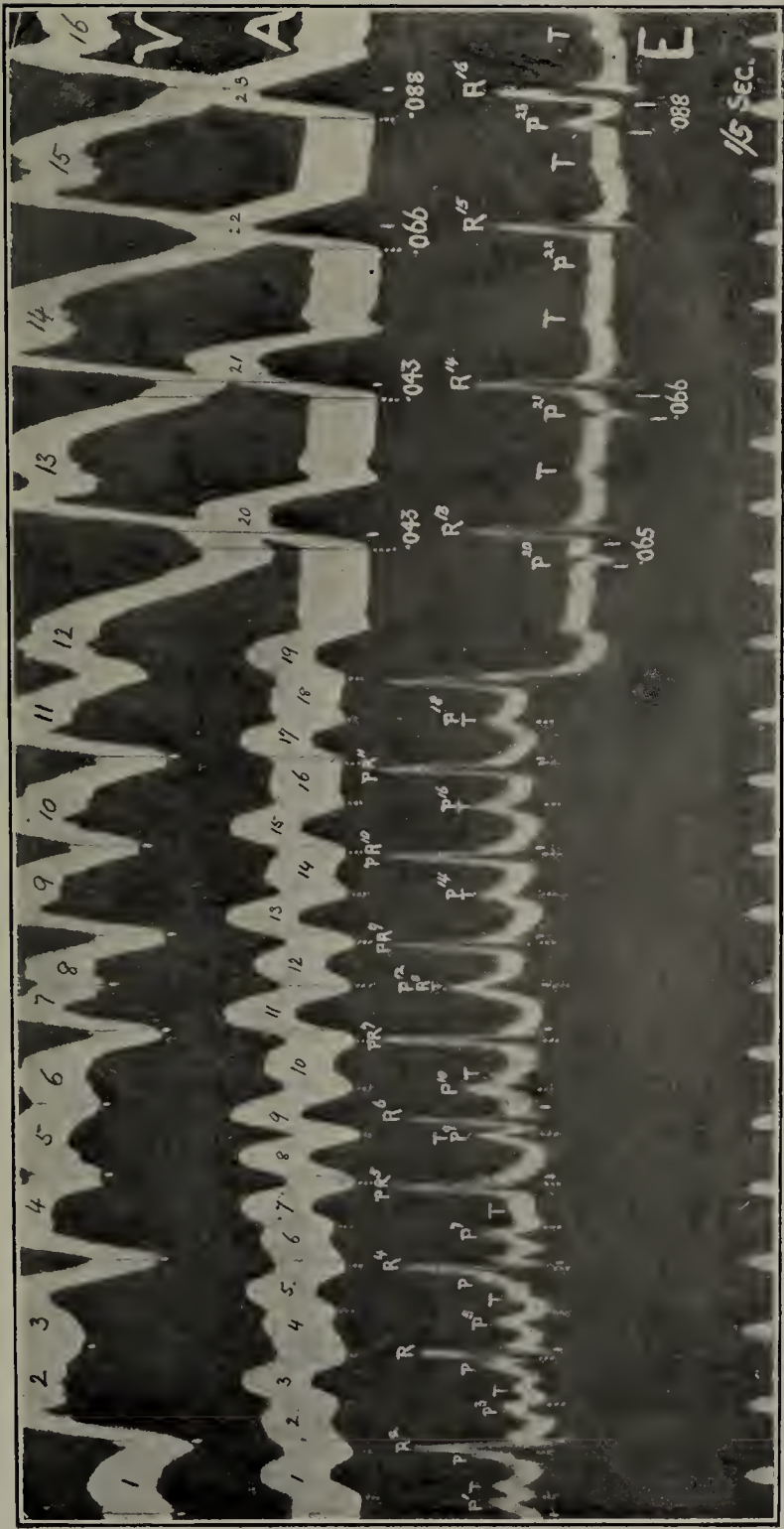


Fig. 144 (*Heart*, 1909-10, I, 306, *Fig. 29*). Simultaneous myocardiographic curves (*V*=ventricle, *A*=auricle) and electrocardiogram from a dog. Over the first half of the figure the auricle was in fibrillation. The break back of the normal sequence is shown, but the first three beats do not arise at the pace-maker. R^{13} and R^{14} are responses to "nodal" impulses, the *P-R* intervals are short and *P* is completely inverted. R^{16} is a response to an impulse originating at the pace-maker. R^{15} occupies an intermediate position, *P* is isoelectric, and the *A-s-Vs* interval (see myocardiogram) is longer than the preceding and shorter than the succeeding *A-s-Vs* interval. For comparison with Plate II, *Fig. 218*.

were also noted (Fig. 47). The auricular representatives of this figure may be compared with the third escaped beat of Fig. 144; in both figures the auricular complexes are isoelectric.

General observations.

The heterogenetic and ectopic rhythms (clinical and experimental, ventricular and auricular) described, present certain features in common. The rhythms are regular and, as shown by a comparison of adjacent beats, spring from single foci; these two qualities are intimately connected with each other. The rate is rapid; in the few instances of tachycardia of ventricular origin, 200 to 210; in the tachycardia of auricular origin, 130 to 240. The onset is abrupt, the change from one rhythm to the other takes place during a single cardiac cycle; the first paroxysmal beat is obviously premature. The offset is equally sudden, and is succeeded by a characteristic "post-paroxysmal" pause.

The examples of tachycardia which are offered should suffice to establish the main propositions postulated. The tranquillity of the normal rhythm may be upset by the interposition not only of single but of successive beats which, so far as the pace-maker is concerned, are of extrinsic origin. There may be a temporary dislocation of impulse formation, and the new rhythm, emanating from a single focus, ultimately sets the pace of, and controls, the rhythm of the whole heart. But the essential feature of the new rhythms of paroxysmal tachycardia is not the site of formation, for there is every reason to believe that no region of the musculature is exempt (instances of their origin in two areas of the ventricle, in the junctional tissues, and in the mass of the auricular tissue have been given); it lies in those characteristics which proclaim such rhythms to be of heterogenetic origin. Their generation from pathological impulses is established by the enhanced rate, and by the peculiarities of the onset and offset, and by their intimate relationships to single premature contractions. They are of cardiac origin, and their causation must be sought in the heart itself; they stand apart from all other forms of tachycardia, in a specific and well defined category.

While we are tolerably acquainted with the mechanism, the further pathology is still wrapped in obscurity. The morbid histology is still an unexplored field. The attacks are the result of heterogenetic impulse formation. The single premature beat arises in similar fashion; and a third condition, to be described presently (auricular fibrillation), is closely related. The elucidation of one will throw much light upon the remaining two. In regard to the factors which directly excite attacks, there are many observations of interest and importance, and none more so than the influence of slight exertion and of posture, where the predisposition to such crises is present. The influence of remedies is capricious, and a slight change of

position, a forced respiratory movement, pressure upon the vagus nerve in the neck, the application of cold to the precordium or the injection of a drug (digitalis, strophanthine) may be followed by instant termination of an attack. Yet where a remedy has proved successful, it may be signally inefficacious upon another occasion, be it a different patient, or the self-same case. And where success has oft attended intervention, the observer is still harassed by a consciousness of the *post* and *propter hoc* alternatives.

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CHAPTER XVI.

PAROXYSMAL TACHYCARDIA (AN OBSCURE FORM;
CLASSIFICATION OF TACHYCARDIAS; RE-
LATIONSHIP TO FIBRILLATION).

An obscure form of tachycardia (paroxysms of regular tachycardia accompanied by the ventricular form of venous pulse).

BEFORE proceeding to a classification of paroxysmal tachycardias, brief attention must be paid to a more obscure form of the affection than those which we have already discussed. The paroxysms give rise to the usual pictures in the arterial curves, sudden accessions of heart rate in which the rhythm is regular. The peculiar feature of the attacks is that they are accompanied by the ventricular form of venous pulse, a form of phlebogram in which no sign of auricular contraction can be found during diastole.^{4, 7 & 8} (Examples of the curves will be found in Fig. 45, 46, 137*b*, and 143.) It is perhaps premature to speak of the mechanism in these cases, as it is by no means finally decided. Yet we have certain definite indications of its nature, and we are aware of certain experimental conditions which give rise to very similar graphic records.

The regularity of the tachycardia speaks for its origin at a single point. The abrupt onset and offset (Fig. 137*b*) and the rate demonstrate that it is of essentially the same nature as those which have been previously considered, namely, that it consists of heterogenetic beats. The absence of presystolic waves excludes it from the category of uncomplicated tachycardias of auricular origin. There are three possible explanations of the mechanism. If an impairment of conduction from auricle to ventricle be supposed, it is possible that the auricular systole of one cycle falls back upon the ventricular systole of the preceding cycle. Under such circumstances *a* and *c* will coincide and the ventricular form of venous pulse will result.¹⁰ Secondly, synchronicity of auricular and ventricular systoles occurs when the two chambers respond to impulses originating between them. In the example given in the last chapter the coincidence was but partial, but there is every reason to believe that it may be complete.² Thirdly, a paroxysm of ventricular tachycardia, retrograde to the auricle, will also give rise to a venous curve of similar form, namely, one in which all the prominent waves fall in the confines of systole (Fig. 134). There is every possibility, nay probability, that each of these mechanisms will find its place in the clinical pathology of the future. But

up to the present time no case has furnished sufficient data to justify its classification under a single one of these headings. It is true that ventricular paroxysms have been observed experimentally and are associated with the ventricular form of venous pulse (Fig. 134); but in the clinical cases, where the ventricular origin of the paroxysm has been established, the venous pulse has not been secured. Again, in the solitary instance in which paroxysms accompanied by the ventricular form of venous pulse have been examined electrocardiographically and in which the end curves were obtained (Fig. 137*b* and 143), there was sufficient evidence of synchronous action of auricle and ventricle and sufficient evidence of the supraventricular origin of the new rhythm,⁷ but no definite decision could be arrived at as to whether the ventricle was responding to auricle or the auricle and ventricle to single impulses formed between them.*

The classification of paroxysmal tachycardia.

The various forms of abrupt and periodic acceleration of heart rate, as they are encountered in clinical work, classify themselves naturally. We may divide the cases into two clear and distinct categories. In the first division we place those examples where the normal heart rhythm, the rhythm derived from the pace-maker, is maintained, and where the impulses are homogenetic or physiological in type. To this class belong all instances of enhanced heart rate as a result of emotion, strain and other causes, where the heart's sequence is normal. We may also include the majority of those tachycardias, where on account of greater susceptibility, either of the pace-maker or of the nervous system, a relatively slight exciting cause produces a profound change of rate. The second division, and the more important, is composed of examples of what may be termed *true paroxysmal or heterogenetic tachycardia*; and in this class two definite groups may be isolated. The first group consists of the tachycardias considered in Chapters XIV and XV; its further subdivision rests upon the location of the points in the musculature at which the new rhythms arise. The second group contains a single entity, namely auricular fibrillation, (Chapters XVII and XVIII).

* In a recent paper, Hirschfelder⁵ has attempted to co-relate these cases with auricular fibrillation. We shall see more fully at a later stage that this position is untenable, if we regard fibrillation as an inco-ordination of the auricle. A regular heart action is never associated with auricular fibrillation if the ventricle is responding to auricular impulses; moreover it may be said of all the cases of regular tachycardia, in respect of which we are in possession of the full data, that they are unifocal in origin. A regular ventricular action implies a similar fundamental mechanism from beat to beat.

A classification of tachycardias, based upon their clinical pathology.

A. *Homogenetic tachycardias*, the result of alterations in the rate of physiological impulse formation at the site of the old pace-maker.

B. *Heterogenetic tachycardias*.

I. Regular tachycardias springing from single foci of the musculature.

a) Arising in the pace-maker (as yet unobserved).

b) Arising in the central regions of the auricular tissue.

c) Arising in the neighbourhood of the auriculo-ventricular node.

d) Arising in the right ventricle.

e) Arising in the left ventricle.

II. Irregular tachycardia probably springing from many foci in auricular musculature.

Auricular fibrillation.

Terminal events in paroxysmal tachycardia; the appearance presented by the auricle in a state of fibrillation.

A paroxysm of regular tachycardia, of the form which we have now fully considered (due to heterogenetic impulses), usually terminates with the resumption of the normal sequence and the re-established dominance of the old pace-maker. Not infrequently however a tachycardia proceeds to a still higher grade of disorder of the cardiac mechanism. We have already noticed, in discussing the experimental phenomena which are observed to follow ligation of a coronary artery, that, as the rate of tachycardia steadily rises until it reaches a point at which the maintenance of circulation becomes critical, the ventricular muscle seems to pass almost insensibly into a condition of absolute inco-ordination or delirium, a final culmination from which in the higher mammalia recovery is extremely rare.

In patients in whom high grades of disorder of the cardiac rhythm are present, unexpected death is not exceptional. And in cases of this kind, where premonitory symptoms fail, it may well be asked (as McWilliam originally questioned⁹) whether death is not the direct result of ventricular fibrillation. We have no definite evidence upon which the question may be decided at the present time. But the close relationship of tachycardia and higher grades of disorder in the ventricle is of importance, for a parallel relationship exists in the auricle; and as the highest grades of auricular disturbance are compatible with continued existence, our knowledge of this relationship in the auricle is necessarily greater.

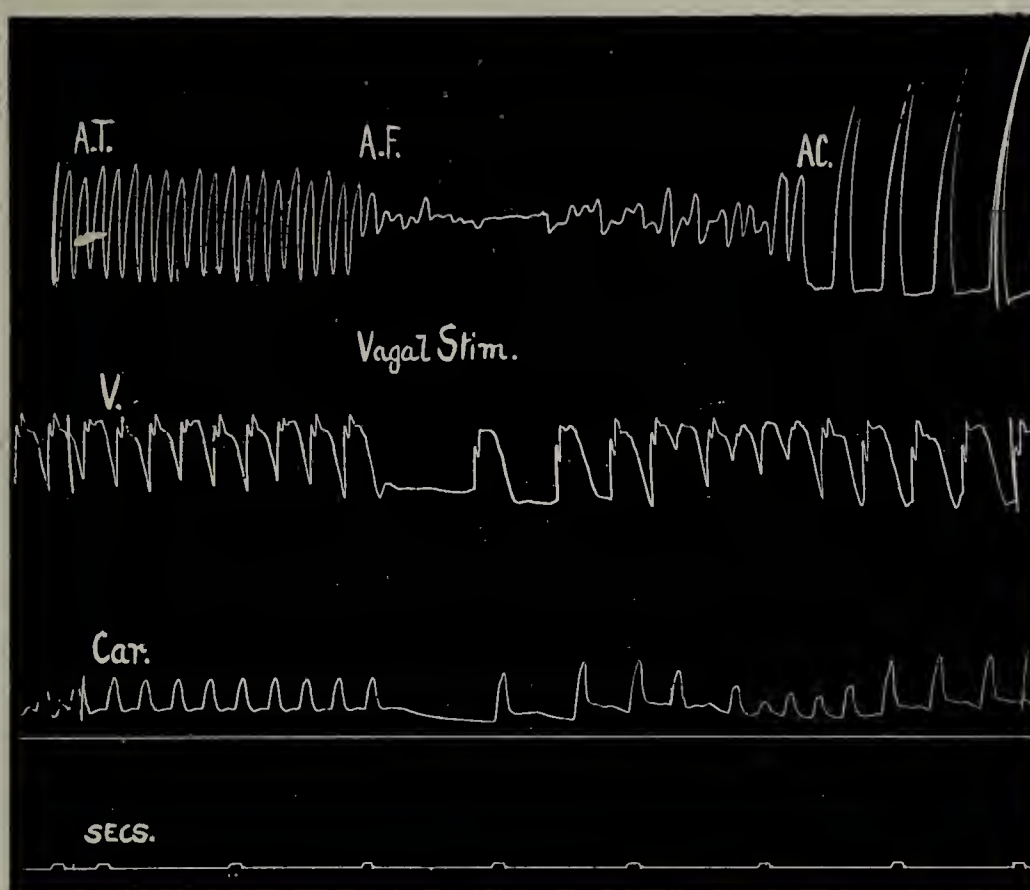


Fig. 145 ($\times \frac{1}{2}$). Myocardiographic curves (*A*=auricle and *V*=ventricle) and Hürthle carotid pressure curve from a dog. At *A.T.* the auricle is responding regularly and at an extremely rapid rate to a faradic current; the ventricle is missing alternate beats. At *A.F.* the auricle passes into fibrillation as a result of vagal stimulation. At *A.C.* the co-ordinate action of the whole heart is resumed. To illustrate the close relationship of tachycardia and fibrillation.

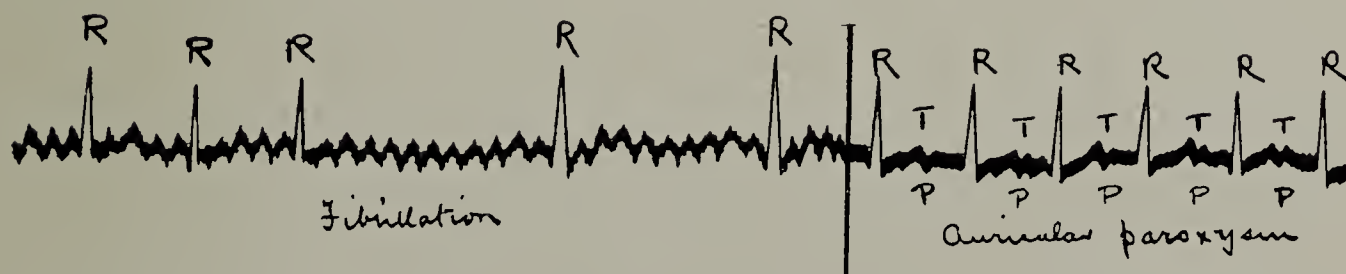


Fig. 146 (*Heart*, 1909-10, 1, 355). A retraced electrocardiographic curve from a patient, the subject of tachycardia of auricular origin. On one occasion abrupt transitions from tachycardia to fibrillation and from fibrillation to tachycardia were noticed. Two inches of curve have been excised at the point where the vertical line is placed. The actual transition was not recorded. To the left the auricle is in fibrillation. To the right it is beating in response to intrinsic and regular impulses arising in a single focus; (Plate I, Fig. 215 is from the same case). The mechanism will be clearer to the reader when he has read the succeeding chapters.

Clinical instances are not wanting in which auricular tachycardias have given place to disturbances, which are attributable to culmination of the auricular disorder. In the next chapters a condition known as *auricular fibrillation* or auricular delirium is described, and this condition is responsible for an extreme or absolute irregularity of the arterial pulse. The passage of a rapid and regular radial pulsation, due to a tachycardia (in all probability of auricular origin) into complete irregularity, has been described by Hewlett,³ and several instances of it, one of which has been recorded, have come under the notice of the writer⁶ (Fig. 145 and 146), (compare Hewlett's recent case⁴).



Fig. 147 ($\times \frac{1}{2}$). Myocardiographic and carotid curves from a dog. At *A.F.* the auricle is in fibrillation, the lever stands in the diastolic position. The ventricle *V.I.* beats irregularly in response to auricle. At *A.C.* the co-ordinate auricular contractions return spontaneously and the ventricular beats resume their regularity (*V.R.*). The fibrillation was induced by faradisation of the auricle.

Fig. 148. A diagram illustrating the author's view of the close pathogenetic relationship of the single premature contraction, the true paroxysmal tachycardia and fibrillation, as they occur in the auricle. The heart is diagrammatised to the right, and the *black* and *red* arrows indicate the general directions taken by the contraction waves which result in response to *homogenetic* and *heterogenetic* impulse formation respectively. In the uppermost pulse curve two premature auricular contractions are shown. In the middle pulse curve a single premature beat and a short paroxysm of tachycardia are observed. In the lowest pulse curve the complete irregularity associated with auricular fibrillation is depicted. The red beats are considered to be of heterogenetic origin. Actual pulse curves have been utilised in the construction of the figure. The view adopted is that the three mechanisms are stages of one and the same process, and that the higher grades of disorder are brought about, either by an increase in the exciting agent or by an increase in the irritability of the tissue responding to it.

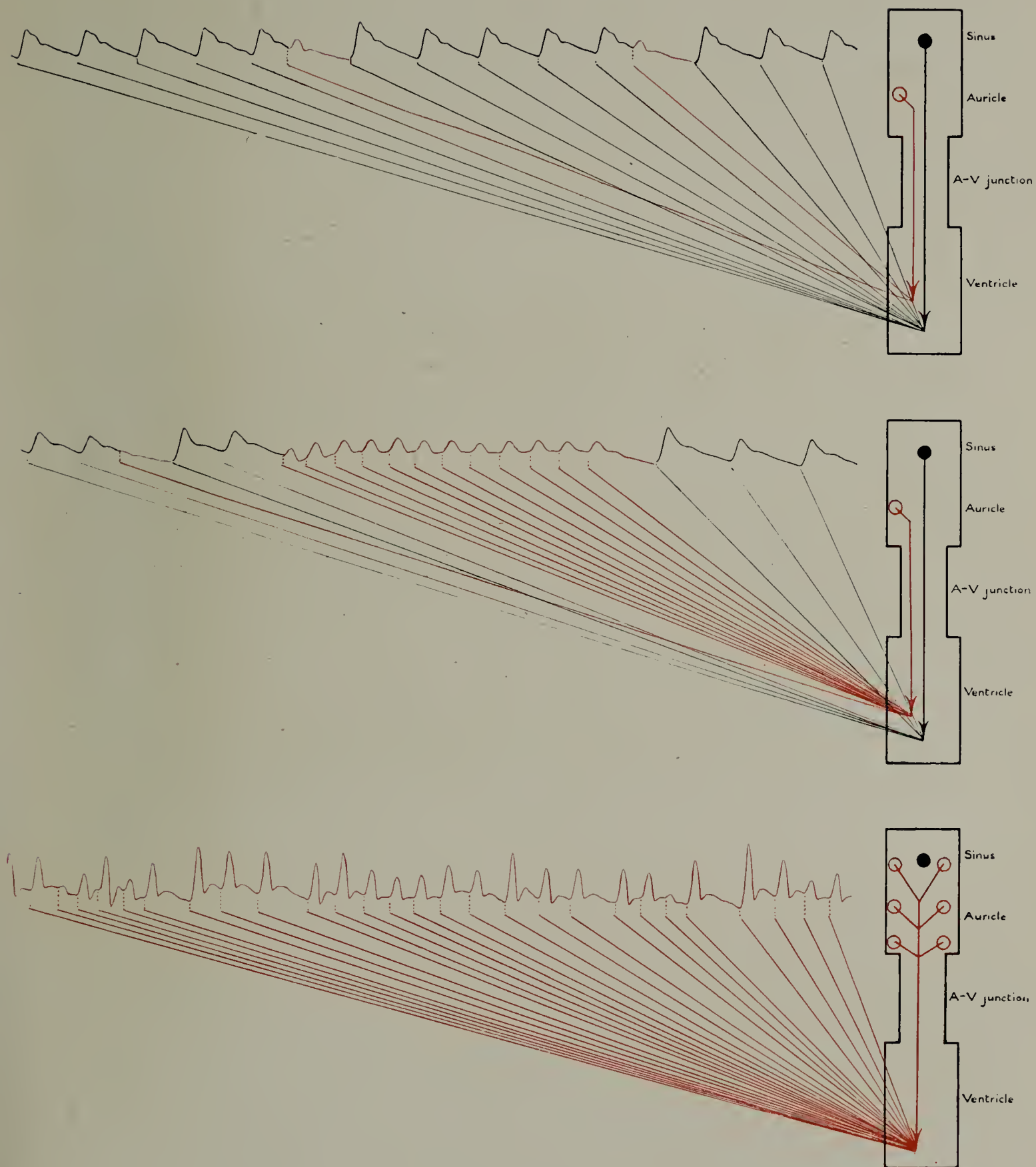


Fig. 148.

The transition from one state to the other may be studied in experiment ; if electrodes are placed upon the auricle and a very weak faradic current is thrown into the muscle, a disorder of the heart's rhythm as a result of premature auricular contractions results. Increase the strength of excitation, and a stage is reached at which the natural pace-maker becomes subservient to a new and regular rhythm arising at the excited point ; the auricle enters upon a rapid and regular tachycardia. Strengthen the faradic current still more, and the rapid and co-ordinate beats of the auricle vanish, sometimes abruptly, often gradually, and the musculature is thrown into a state of delirium. When the auricle is contracting at a high speed in response to intrinsic and pathological impulses, it is ever on the verge of delirium. The two conditions, whether they are produced artificially or arise spontaneously, are intimately connected, and it is difficult if not impossible to avoid the conclusion that there is a close association between the origin of the one and the other.

In the preceding chapters we have passed from a description of single heterogenetic impulses to a consideration of successive impulse formation from a single focus. May we not anticipate that under given conditions impulses are simultaneously generated in many foci in the auricular tissue ? And if such a phenomenon exists, to what condition will it give rise ? The contraction waves, starting in many places, will meet or collide in the auricular tissue, a co-ordinate contraction will be impossible, for the systole of each area will be imperfect and synchronous with the systole or diastole of surrounding muscular areas.

Now the actual features of auricular delirium as they are visible to the naked eye, and in an experiment, are as follows¹¹ : the walls of the auricle stand in the diastolic position (Fig. 147) ; systole, either complete or partial, is never accomplished ; the wall as a whole is stationary ; but careful examination of the muscle reveals an extremely active condition, it appears to be alive with movement ; rapid, minute and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface. Mechanical records from the tissue have little or no excursion. This is the condition which is properly termed *fibrillation of the auricle or auricular delirium*. And the explanation of the provocative cause is that which has been spoken of. There is reason to believe that the auricular tissue as a whole is broken up into a lesser or greater number of small areas, each of which is independently and spontaneously elaborating heterogenetic impulses.¹ At times the movement is coarser, and then mechanical records usually discover an attempt at regularity. Thus, just as in the ventricle a rapid tachycardia passes imperceptibly into a fully developed delirium, so in the auricle the actual transition point from tachycardia to fibrillation, though it may be abrupt, is often quite indeterminable. The view of the relationship of the mechanism, where single premature beats, tachycardia of ectopic origin and finally fibrillation exist, is expressed in the accompanying

diagram, in which actual arterial curves from the respective conditions are incorporated, (Fig. 148).

In the succeeding chapters we shall speak more fully of the condition known as auricular fibrillation, and it will be our special endeavour to place the proof of its occurrence in man as clearly before the reader as possible.

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CHAPTER XVII.

CLINICAL AURICULAR FIBRILLATION.

HISTORICAL.

IN the following pages an account is given of a specific clinical condition, characterised in the great majority of cases by complete irregularity of the arterial pulse and by an absence of all signs of the normal auricular contraction.

The irregularity, which is one of the chief features of the condition, is the commonest persistent irregularity exhibited by the human heart, constituting as it does approximately 50 per cent. of all such cases ; it will be demonstrated that the disturbance of cardiac rhythm is to be sought in the auricle and attributed to temporary or permanent inco-ordination of the musculature of this chamber.

Our acquaintanceship with the facts upon which this final conclusion rests is the outcome of the studies of a large body of workers. A retrospective survey of the observations upon clinical material permits the recognition of cases, belonging to the category discussed, over a period of many years. Fully possessed of the facts, we may trace the earlier descriptions of the condition along two lines ; independent and distinct observations were carried out upon either the arterial or the venous systems. The two paths have converged and finally have met during the last decade.

On the one hand a markedly irregular arterial pulse, especially associated with mitral disease in its later stages, was the subject of study by mechanical means from the time of the introduction of the sphygmograph. It is portrayed by Marey,³² Riegel,³⁷ Sommerbrodt⁴⁵ and many other writers. It has been termed the "mitral pulse," and has been attributed to "delirium of the heart" amongst other causes. It has passed by the names *Pulsus arrhythmicus* and *Pulsus irregularis*,³⁷ and has been identified, in a classic but obsolete nomenclature, with the adjectives *irregularis*, *inæqualis*, *deficiens* and *intermittens*.

On the other hand, a prominent systolic pulsation in the veins of the neck was described,^{1 & 44} and was attributed, and has long since been held

as due, to tricuspid incompetence. The subjective timing of the venous pulsation was endorsed by Riegel, who obtained the first graphic records of the movement; but the class of cases in which such pulsation is found essentially was not isolated, neither was its full significance grasped, until the more exact and more applicable technique of Mackenzie was introduced.

It is since the year 1902, when the *Study of the Pulse* was published, that chief progress has been made. To Mackenzie we owe the definite correlation of the two phenomena, gross irregularity of the heart and the systolic venous movement, which he has termed the "ventricular form of venous pulse." In the work referred to, this writer first demonstrated their frequent association and ascribed them both to a single underlying condition, namely paralysis of the auricle. A year later Hering,⁸ describing the arterial pulse alone, laid more stress upon its characteristics and spoke of it under the title *pulsus irregularis perpetuus*. The recognition of its specificity has been arrived at gradually and the facts supporting the conclusion have been contributed largely by Mackenzie. In particular, the emphasis which he laid upon its frequent association with the ventricular form of venous pulse in 1904,²⁷ and the prominence given to this observation in a later paper, based upon an examination of 500 cases,²⁸ must be noticed. But in reality it is only since the galvanometric examination of the heart has been available that the probability of its specific nature has grown to certainty.

In his papers of 1904-5, Mackenzie brought forward several new and important facts, and most striking amongst them, in the light of our present knowledge, were evidences that the auricle is active. Formerly regarding the auricle as paralysed, because no sign of activity could be found, he now attempted to separate a special group of cases in which auricular activity might be considered probable. Auricular activity was assumed, because the auricle was found hypertrophied at autopsy; and because certain instances were observed in which the normal rhythm reasserted itself. It is to these papers that we are more especially indebted for the observation that in all cases of complete irregularity of the heart there is an entire failure of signs of the *normal* auricular contraction during diastole; and further, for the first record of cases of this nature, in which it is probable that little dilatation of the right heart and little tricuspid regurgitation is present. His earlier view that the condition results from auricular distension as a consequence of valve incompetence was at least partially abandoned, and the rhythm was ascribed as the cause rather than the result of the eventual dilatation. In 1904 Mackenzie postulated the view that in many cases the seat of the rhythm is in the junctional fibres lying between auricle and ventricle, and by conceiving the simultaneous contraction of auricle and ventricle in response to impulses from this single source, he attempted to explain the absence of every sign of normal auricular contraction which he had demonstrated to be one of the chief features of such cases.

In 1907-8 Mackenzie^{29 & 30} adopted the hypothesis of the nodal origin of the rhythm more generally, holding the auriculo-ventricular node to be the seat of disturbance in all cases of complete irregularity found in combination with the systolic form of venous pulse. He therefore included all such cases under the term "nodal rhythm."

We have seen in a preceding chapter that true "nodal rhythm" is a very definite entity; the condition is extremely rare. The view that the irregularity under consideration is of this nature has been abandoned by Mackenzie; the reasons for its abandonment are set forth in the succeeding pages.

The description of the condition as set forth by the writer is based upon a detailed examination of 106 cases.

THE CLINICAL CONDITION.

The radial pulse curves.

The character of the radial pulse curves in complete irregularity of the heart is so striking that it could not, and as we have seen did not, escape early attention.

The irregularity is of the most varied description. The pulse may be slow or fast, and the variation in rate is great (30 to 200). All the beats may be of small excursion; more commonly there is a haphazard intermingling of forcible and weak contractions, and the latter are often markedly dicrotic. The radial pulse is but an indifferent index of the rate of the ventricle; many beats are not transmitted. The pulse rate may be considerably reduced, either as a result of these dropped beats or as a consequence of the actual slow speed of the ventricle. The beats may show coupling over short or long stretches of curve. The fast types are the commonest, and in these the usual rate of the ventricle is approximately double the normal rate (110 to 150). It is usually at these fast rates that the disorderly character of the pulsation is so prominent. With the slower rates the irregularity is less marked; nevertheless, it is always present, a fact which can be determined by careful measurement of the tracings. In arterial curves the disorder may be recognised by two criteria. First and most important is the absolute character of the arrhythmia. The heart action is never regular, and seldom or never do two beats of the same character or length succeed each other. In a long curve, it is rare to find any two short sections of tracing which have even a superficial resemblance to each other. The pauses betwixt the beats bear no relationship to one another, and in this feature the irregularity stands in marked contrast to all other varieties. The second criterion consists in the absence of a definite and continued relationship between the strength of a beat and the length of the pause which precedes it. A strong beat may follow a short



Fig. 149 ($\times \frac{5}{8}$). (*Heart*, 1909-10, 1, 315). Radial pulse curves taken with a Dudgeon sphygmograph. The time tracing, which applies to all curves beneath it, is in $\cdot 2$ sec.. The figure illustrates the general characteristics of the disordered heart action considered in the text.

- 1 and 2. From a man aged 48, admitted to hospital suffering from mitral stenosis of rheumatic origin and general cardiac dilatation. Enlargement of liver, distended veins and dropsy were present. Irregularity complete and persistent; murmurs early and mid-diastolic; venous curves of ventricular form; electrocardiographic curves of usual type. The oscillations were maximal when the electrodes were placed over the right auricle.
3. From a man aged 64, the subject of bronchitis, emphysema and arteriosclerosis. No history of rheumatism. Heart somewhat enlarged to right and left. Heart sounds normal; S. B. P., 150 mm. Hg.* With the exception of shortness of breath on exertion no signs of broken compensation were present. The irregularity disappeared on one occasion for a few days, the pulse regularity was then interrupted by auricular extrasystoles. The *a-c* interval was normal. With the complete irregularity the venous pulse was ventricular in outline, the electrocardiograph was typical, the oscillations were maximal in leads from the parts of the chest wall covering the right auricle. There was no pulse slowing with heavy doses of digitalis.
4. From a man aged 37, suffering from mitral stenosis of rheumatic origin. Heart enlarged to right and left, dyspnoea and slight liver enlargement, no dropsy. Pulse persistently irregular; ventricular form of venous pulse; electrocardiograms typical; oscillations maximal with electrodes in neighbourhood of right auricle.
5. From a man aged 65, suffering from aneurismal dilatation of the whole thoracic aorta, pulmonary oedema, associated with arterial sclerosis, emphysema and signs of sclerotic kidney. Dropsy and liver enlargement present. Pulse persistently irregular; ventricular form of venous pulse. Died unexpectedly.

* This blood-pressure reading is a measure of the obliteration pressure of the most forcible beats. Blood-pressure estimations in cases of complete irregularity are extremely unsatisfactory; the beats force their way through the armlet at widely varying pressures

pause, and a weak beat may succeed a long pause. A few examples of the pictures presented by Dudgeon tracings are given in Fig. 149. They may serve, with the brief notes attached to them, as a guide to the recognition of the type of case with which we are dealing. They illustrate the main points referred to in the text, but the variety shown is so great that they can scarcely be held even as representative of the irregularities which may occur. Numerous and additional examples are scattered throughout the simultaneous tracings which illustrate this and the succeeding chapter.

The venous pulse curves.

“The ventricular form of venous pulse” is a term which expresses the only fixed quality manifested by graphic records taken from the jugular veins in these cases. It implies that all prominent and rapid changes of volume in the venous cistern fall within the limits of ventricular systole. The curves corresponding to the individual heart beats vary in their positions relative to each other just as do the radial beats. There may be considerable variation in the amplitude of the separate curves in a given case. This variation is far from instrumental in origin, for close examination reveals the recurrence of a particular type of curve with a given length of pause, a given type of radial beat or a given phase of respiration. As a general rule, and in a single case, a large venous curve accompanies a large radial curve, but the difference in size from one beat to the next is less in the former than in the latter. A family resemblance between the separate venous beats of a single curve is generally if not always present.

The complete curve, corresponding to a single heart cycle, is generally composed of two or three peaks, and a similar number of dips. The upstroke of the first peak is synchronous with the commencing carotid pulsation at the same level of the neck (though it may precede or succeed it slightly). The downstroke of the last peak starts at a point corresponding to the opening of the tricuspid valves. It is synchronous with the bottom of the downstroke of the cardiogram, or with a point a little later than the bottom of the dicrotic notch on the carotid tracing. The chief depressions follow the first and last peaks and are very variable in degree from case to case. As a general rule it may be said that the shorter the duration of the abnormal mechanism the deeper is the first as compared to the second depression; and that in old-standing cases the dip in the centre of systole is replaced by a larger and fuller complex of systolic peaks. A definite relationship appears to exist between the mean distension of the veins and the swelling of those veins in systole. Thus, in cases of long duration, in which the veins are more or less markedly dilated, the venous curve is in the form of a prominent systolic plateau. The older conception, that the prominence of the venous pulsation is an index of the degree of tricuspid reflux, is not without a definite foundation. The curves obtained from

patients soon after the onset of the new rhythm, and the curves in cases in which the right heart engorgement is small in degree, generally permit of close comparison with the *ventricular* portions of the venous curves taken from normal subjects (the parts of the normal curves marked *c* and *v*). A curve of the kind is shown in Fig. 150 *C*. In long standing cases, or in instances where compensation is less complete, the first depression (corresponding to the *x* and *x'* dips of the normal curve) is filled (Fig. 150 *A* and *B*), and the filling may happen in greater and greater degree until the type assumed is flat-topped and resembles the curve of intra-ventricular pressure (Fig. 150 *A* and 152 *A*). The transition from one type to the other may be followed from case to case, or may be seen in one and the same case as cardiac tone and venous filling wax and wane. The flat-topped type also consorts more commonly with rapid heart action, though in marked degree it rarely occurs in the absence of appreciable heart distension. In many curves the smaller and more rapid beats are accompanied by the flat type of curve, and the stronger beats by a bifurcated type (Fig. 150 *A*). The contrast may be explained by the unequal heart filling under the two conditions. The accompanying figure illustrates several of the types of curve met with, and others will be found in this and the succeeding chapters. It is by no means unusual to see three prominent peaks, concurrent with each heart beat (Fig. 150 *D*).

The relationship of the normal type of curve to the various forms of ventricular venous pulse curve is diagrammatised in Fig. 151. The outlines have been drawn from actual curves. The dotted outline is that of the auricular form of venous pulse. The diagram displays transitions between various types of the ventricular form of venous pulse. But the distension of the auricle and the deformation of the venous curve may take place, while, during the transition, the presystolic auricular contraction is present, or while, during the transition, the co-ordinate systoles of the auricle are suspended. The ventricular form of venous pulse may be conspicuous even in its plateau form, while the normal heart sequence is maintained (p. 62), and, as we have already seen, the usual or normal systolic portion of the venous pulse may be found (the type with the deep *x* and *x'* depressions) and yet the signs of co-ordinate and presystolic auricular contraction may be entirely in abeyance. It should be clearly comprehended that two perfectly distinct phenomena exist, a sudden disappearance of all signs of the normal auricular systole, and a gradual increase of pressure in the right side of the heart. The lack of due appreciation of this essential distinction in the past has led to many misconceptions of the condition with which we are dealing, and to the faulty interpretation of many curves. The diagram may stand as an example of the transition of the systolic portion of either auricular or ventricular form of venous pulse curve from the type with perfect to the type with imperfect venous flow. In brief, it may be said that there is but one distinctive quality between auricular and ventricular forms of venous pulse, and it consists in the absence of

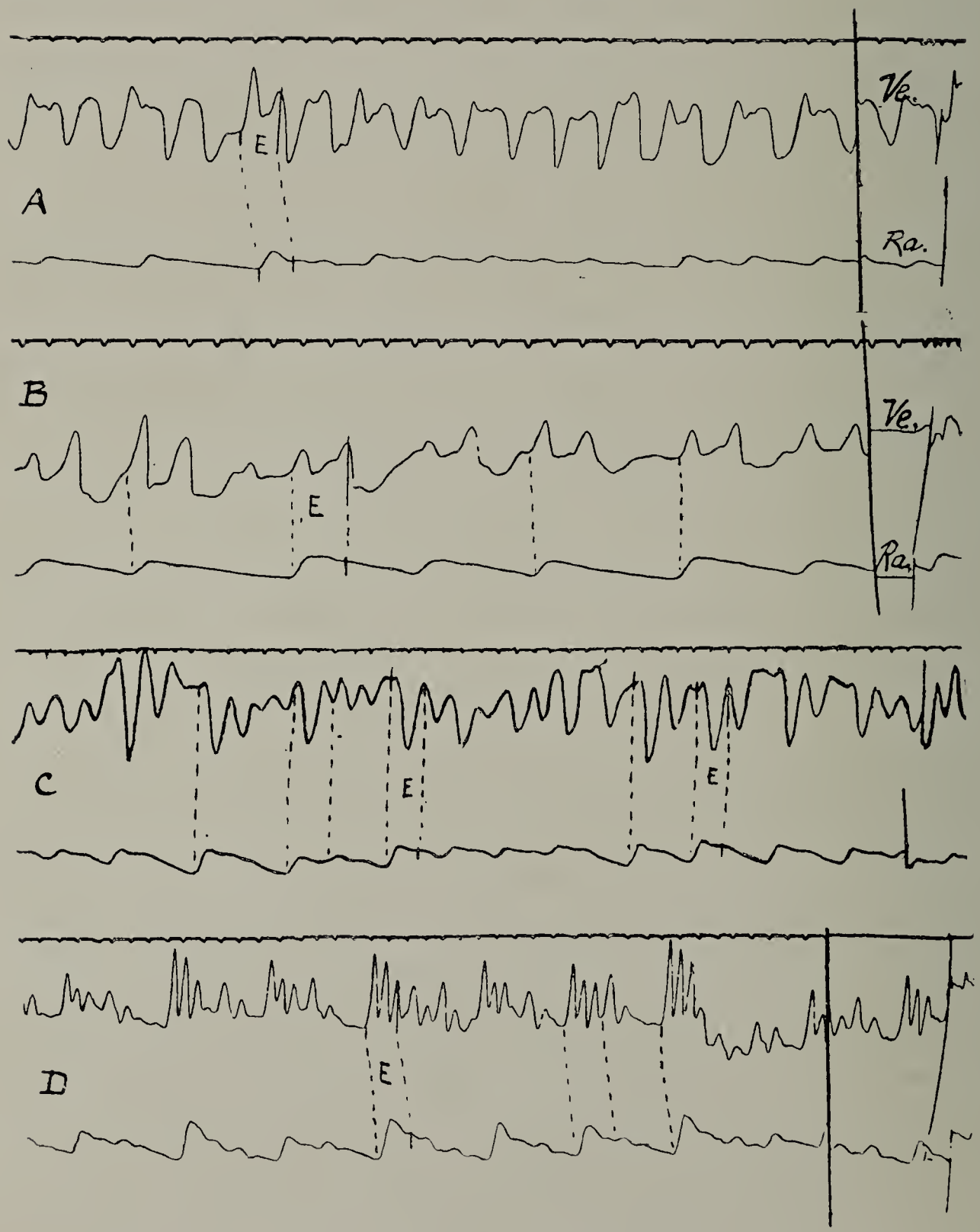


Fig. 150.

Fig. 150 (*Heart*, 1909-10, 1, 318). Simultaneous venous and radial curves from four patients presenting complete irregularity of the heart and the ventricular form of venous pulse. The figures illustrate some of the different types of venous curve met with in this condition. The venous curves are constant in that all rapid upstrokes lie within the confines of systole (*E, E*). The dotted lines unite points representing one and the same phase of the cardiac cycle. The heavy vertical lines cutting a whole tracing mark the points where curve has been excised.

- A. A man aged 39. The mother had rheumatic fever. Admitted for shortness of breath and precordial distress. Heart enlarged to right and left, veins prominent and liver dullness increased. No dropsy. Heart sounds weak but otherwise normal. Electrocardiograms typical, oscillations maximal over auricle. Four inches of curve have been excised from this figure.
- B. A woman aged 23, the subject of mitral stenosis of rheumatic origin. Admitted with enlarged liver and dropsy. The curves were taken some weeks after admission, when the patient had responded well to digitalis. Early and mid-diastolic murmurs; electrocardiograms typical. Eight inches of curve excised.
- C. A man aged 26, with no symptoms other than slight short-windedness on strenuous exertion. History of rheumatic fever. No cardiac enlargement, no murmurs. Irregularity has been present for two years and has been persistent. The electrocardiograms show prominent oscillations and premature ventricular contractions. (Plate IV, Fig. 222 is from this case.)
- D. A woman aged 38, suffering from mitral stenosis. On admission dropsy and œdema of lungs were present. Tracing taken several months later, subsequent to digitalis. Compensation had so far improved as to allow her to pursue her ordinary duties as housewife. Heart enlarged to right and left; early and mid-diastolic murmurs. Veins not prominent. Irregularity persistent. Two inches of curve excised.

the auriculo-systolic or *a* wave from the latter. Individual cycles of a curve, which is an example of the ventricular form of venous pulse, can be identified as such with certainty only by attention to this fact.

Certain waves which occur in diastole.

It has been stated that the ventricular form of venous pulse is composed of waves of which the most prominent and abrupt fall during systole. It is also true that waves occur within the limits of diastole.

We have seen that, apart from the auriculo-systolic wave, there is no essential distinction between the individual venous curve in the normal and abnormal conditions. It will be necessary at a slightly later stage to note a single exception to this rule. But, for the time being, we may enquire whether any waves occur in the diastolic reaches of the normal venous pulse, apart from those allotted to auricular movement. There are waves of two kinds, and precisely similar waves are found during diastole in the condition which we are considering. There is a wave which

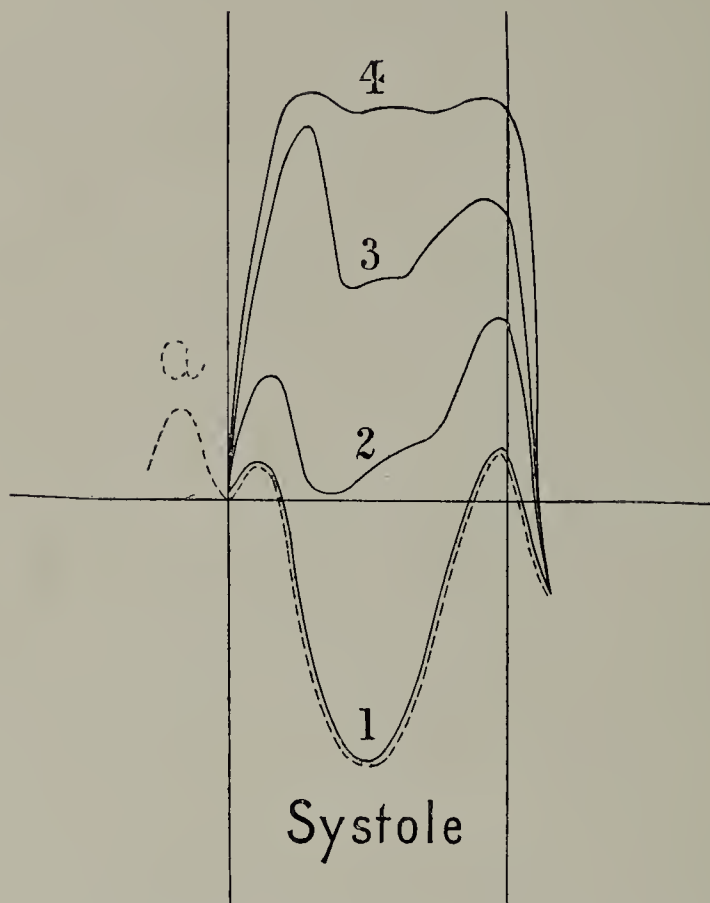


Fig. 151. A diagram illustrating the relationship of the auricular and ventricular forms of venous pulso, and the more common variations in shape to which they are liable. The dotted line represents the usual or physiological type of the auricular form of venous pulso. The continuous lines represent the ventricular portions of types of curve met with in auricular and ventricular forms of venous pulse.

Fig. 152 (*Heart*, 1909-10, I, 323). Simultaneous venous and arterial tracings from three patients with complete irregularity of the heart. Illustrating the several types of wave which occur in diastole, when the venous pulse is ventricular in form.

- A. From a patient the subject of old-standing mitral stenosis. The curve shows fibrillation waves, *fff*, during the long diastolic pauses. These are superimposed upon long stasis waves. The systolic portion of the venous curve is of the plateau form.
- B. From a case of mitral stenosis. There is a wave in diastole marked * which is ascribed to closure of the tricuspid valves at the end of filling. In the longest pause small oscillations, *f*, appear.
- C. From a man aged 49, suffering from mitral stenosis of rheumatic origin. The heart considerably enlarged to right and left. Dropsy and pulsatile liver. Early diastolic murmur at apex. The pulse is completely irregular, except that each large beat is followed by a smaller one at an almost constant interval. In the diastolic portion of the curve a gradual rise ends in a plateau, and where the two meet three small oscillations occur (marked *x*). They were accompanied by a valvular sound at the apex and in the neck. They may be attributed to closure of the tricuspid valves.

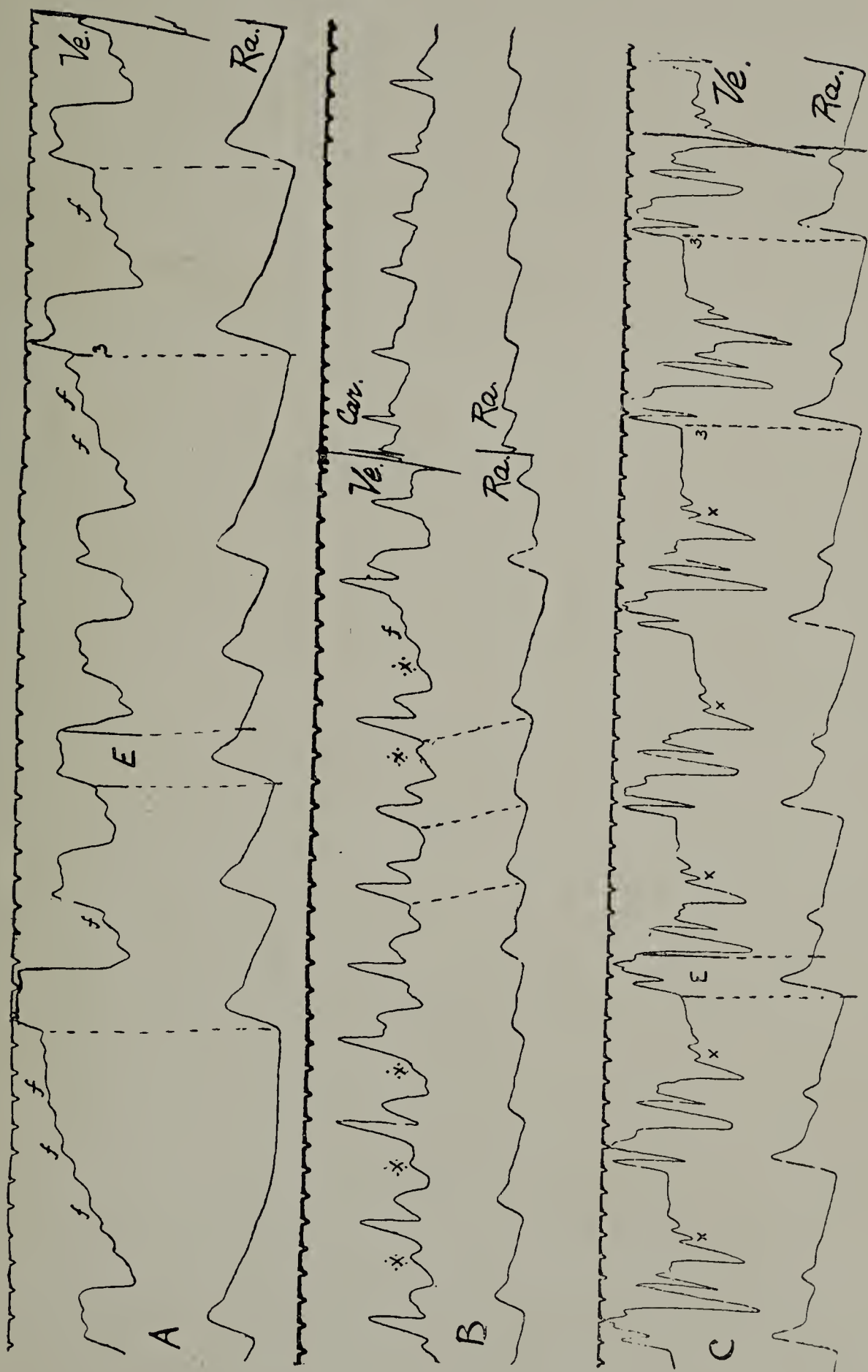


Fig. 152.

has been termed³³ the "second onflow wave," and its causation is assigned to the overflow into the veins when in a long diastole the heart is already full, or to the increase of pressure in the ventricle during the filling. It is seen to advantage in the accompanying figure (Fig. 152). At the commencement of tracing *A* there is a long pause, and during the whole of it the veins are steadily swelling. It is also prominent in tracing *C* and is shown to a less extent in tracing *B*. The fixed relationship of this stasis wave to the preceding systole and the variation of its extent with the length of diastole permit its recognition.

The second type of wave was first described in the normal condition by Hirschfelder,¹⁴ and has been attributed to the closure of the tricuspid valves at the end of the ventricular filling.⁶ Waves belonging to this class, and termed *h* or *b* waves, are seen in Fig. 152 *B* and *C*. In tracing *C*, and just at the summit of the main sweep of the stasis wave a triple vibration (marked \times) with constant time relationship to the preceding systole is plainly visible. In tracing *B* we have an example in which the portions of the diastolic waves attributable to stasis and valve movement are more difficult to unravel. The longest diastole, just antecedent to the stops, is of value in this connection. In this diastole we see the general sweep of the stasis wave and superimposed upon the first part of it is the wave marked \times . The same wave is found in earlier portions of the same curve, and the comparison with the later diastole allows us to gauge the extent to which stasis aids in its production in these earlier diastoles.

We may now turn to another type of diastolic wave, the most important of all. It forms the exception to the remarks previously made as to the resemblance between the ventricular and auricular types of venous curve. These undulations are only met with as an accompaniment of the ventricular form of venous pulse. They are seen but occasionally, and only when the pulse is slow.⁴⁶ They are well marked in tracing *A*, and are more obscurely figured in tracing *B* (marked *f*). They are also shown in the photographic curve (Fig. 168). They are multiple, and the rate may be from 350 to 500 per minute. Their irregularity renders it difficult to estimate the rate other than approximately. They were first described by Wenckebach.⁴⁶ Too much emphasis cannot be laid upon the fact that complete irregularity in the arterial curve is *never* associated with the auricular form of venous pulse. The waves which occur in diastole can always be shown to arise from causes other than co-ordinate contraction of the auricle, (compare the papers of Magnus Alsleben³¹ and Pletnew³⁵).

The electrocardiographic curves obtained by leads from the right arm and left leg.

Electrocardiographic curves from cases of complete irregularity of the heart have been published by a number of writers.^{5, 11, 15, 17, 19, 22 & 39}

The following account is based upon curves taken from sixty cases.

Firstly, the curves consist of tall peaks *R*, corresponding to the commencement of ventricular systole, and scattered throughout in irregular profusion. Their arrangement is obviously due to the arrhythmic action of the ventricle (*cp.* Fig. 154 and its accompanying radial curve). The direction of the electric variation, of which the peak *R* is an expression, indicates negativity of the arm electrode, or primary activity of the base of the heart. The variation is the same as that which occurs in the normal subject and is an evidence that the ventricular contraction commences at its normal starting point. The remainder of the curve and its comparison with the normal justifies this conclusion, as we shall see in the sequel.

A comparison of the height of the peaks *R* with the strength of corresponding radial beats in synchronous tracings reveals the fact that there is no fixed proportion between them (Fig. 154).

The ventricular summits *Q* and *S* may also be seen in certain curves; the *T* variations are also found. The latter are often very obscure, and this obscurity is the result of the presence of certain special oscillations upon the curves. The variation *T* is plainly perceptible in Fig. 153 *III* and *IV* and 158 *I*, while it is overshadowed in Fig. 153 *I* and 154. At times it is quite as prominent and almost as regular from cycle to cycle as in the normal curves (Fig. 170). The occurrence of *T* is of importance because it demonstrates what would otherwise be uncertain, that the ventricular contraction starting at the base pursues its usual path in the ventricle.

The most striking feature of the electric curves is the absence of all sign of the regular presystolic variation (*P*), which accompanies all normal heart beats, and the presence of that which replaces it, namely a number of irregular oscillations varying in form and prominence (Fig. 153). In leads from arm and leg these oscillations are often marked in amplitude, and the latter may equal if it does not exceed that of the usual auricular variation *P*. In other curves they may be far less conspicuous, and may be distinguishable only from place to place (Fig. 153 *III* and *IV*, *ff*). They are a constant feature of cases of complete irregularity of the heart (if not in one lead then in another) and occur in no other condition. They are responsible for the distortion or concealment of *T* in the arm-leg leads; the clean cut character of *R* is never affected, and this is due to the quickness of the movement. The oscillations appear in a purer form when certain special leads are adopted, but the curves obtained in this way will be more appropriately dealt with subsequently.

The oscillations are most conspicuous when the pulse is slow or during a diastole of unusual length. For during systole they fall upon the ventricular elements of the curve and their definition is obscured. When the heart-beat is rapid the individual waves may be difficult to separate, but their presence is known from the fact that the *T* waves vary greatly in form or are entirely overshadowed (Fig. 154), and by the appearance here and there of single undulations on the curve which can

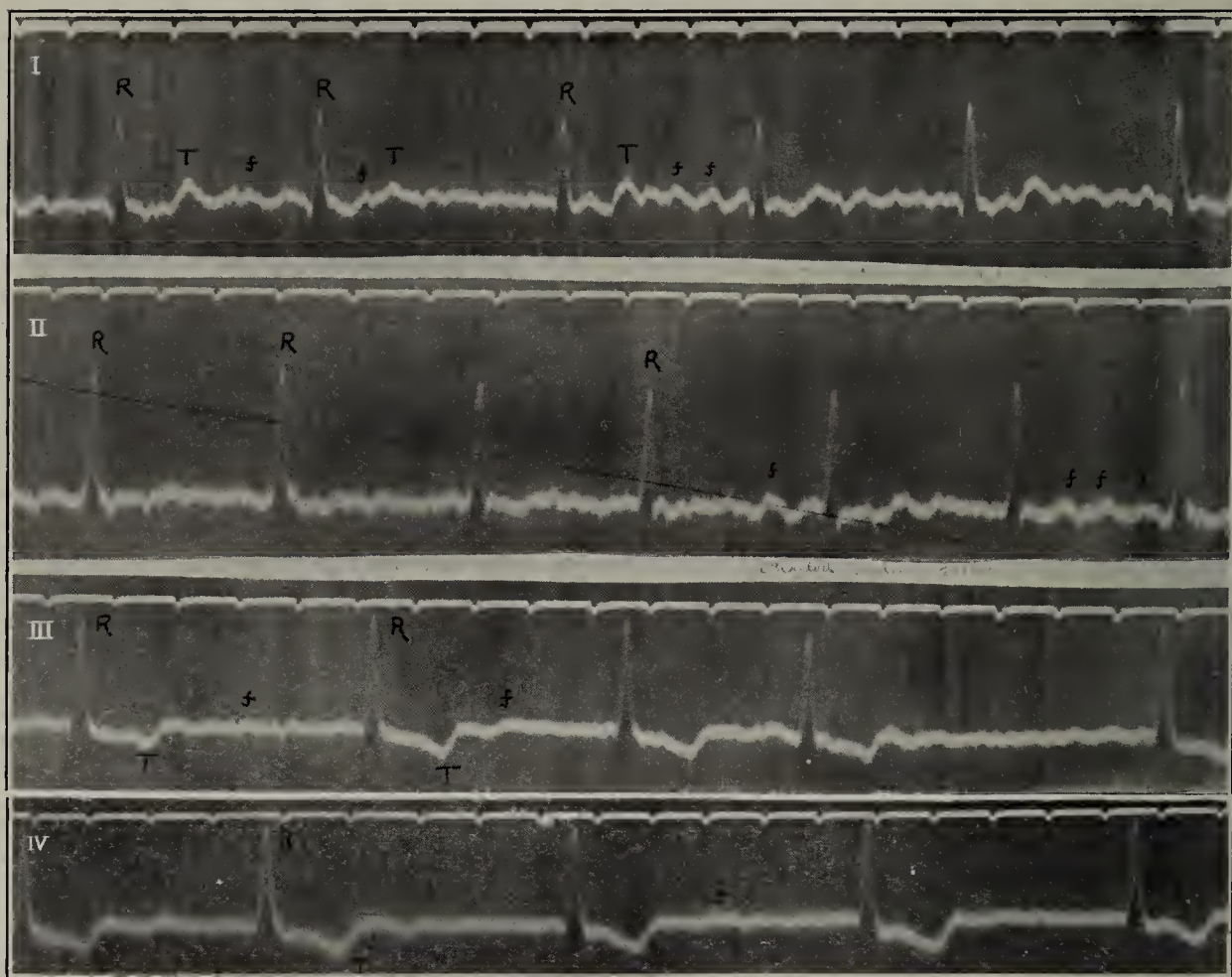


Fig. 153 ($\times \frac{5}{9}$). Four electrocardiograms from patients the subjects of auricular fibrillation. Showing the absence of *P* variations, the complete irregularity of the ventricle and the supraventricular origin of the impulses. The oscillations are variable in their amplitude, but are quite distinct in all the curves.

- I. From a man aged 23, suffering from mitral stenosis. The pulse had been irregular and the ventricular form of venous pulse had been present for two months only. Patient is since dead.
- II. From a man aged 56, who had shown irregularity of the pulse for 10 years. The venous pulse was of the ventricular form. No history of rheumatism. No valve lesion.
- III. From a woman aged 41, suffering from aortic regurgitation, mitral stenosis etc.. Irregular pulse, venous pulse of ventricular form. *T* is inverted.
- IV. From a man aged 53, admitted with arteriosclerosis, ascites and dropsy. *T* is inverted.

be ascribed to no other cause. The deformity of the curve produced in this manner, is as a whole, so characteristic of the condition that, once recognised, the curves are never mistaken for those obtained in any other affection.

COMPLETE IRREGULARITY OF THE HEART IS THE RESULT OF
AURICULAR FIBRILLATION.

The irregular oscillations seen upon galvanometric curves are due to an inco-ordinate contraction of some portion of the heart; they are not a direct result of structural change in the heart, and are independent of movements of the somatic musculature.

It might be suggested that the oscillations in question are dependent upon structural changes in the heart muscle, and that the propagation of the contraction wave, along the path usually taken, is hindered or modified by the presence of areas of tissue affected by disease. But we are in a position to deny that the oscillations are attributable to this cause.

Such a hypothesis is at once weakened when it is known that the oscillations bear no relationship in their extent or frequency to the signs of damage of the myocardium. For a patient may be suffering from obvious and gross myocardial change, yet if the sinus rhythm is dominant the oscillations are absent; and, on the other hand, patients showing no signs of gross myocardial affection, patients in whom there is no dilatation and in whom there is little disturbance of the circulation even after strenuous exercise, exhibit fully developed oscillations, provided that the pulse is completely irregular. If further evidence is required it will be found in cases in which a comparison of the normal and abnormal mechanisms in the same subject is secured.

The curves shown in Fig. 154 and 155 were secured within two days of each other and from a single patient. Fig. 154 has no *P* variations, the heart beat is absolutely irregular, and oscillations are present. Fig. 155 shows the reappearance of the *P* variations, when the pulse returns to its slow and regular rhythm; the oscillations have vanished.

Polygraphic curves from another case are shown in Fig. 156. They were taken within 12 days of each other and present examples of the normal rhythm, interrupted by premature auricular contractions (Fig. 156 *A*) and complete irregularity accompanied by the ventricular form of venous pulse (Fig. 156 *B*). Electrocardiograms taken on the same days are shown in Fig. 157 and 158. The first figure has been described on page 141; it shows prominent *P* variations in a normal rhythm, interrupted by premature auricular contractions. The second, Fig. 158 *I*, is an electrocardiogram from right arm and left leg; *P* has vanished and is replaced by oscillations.

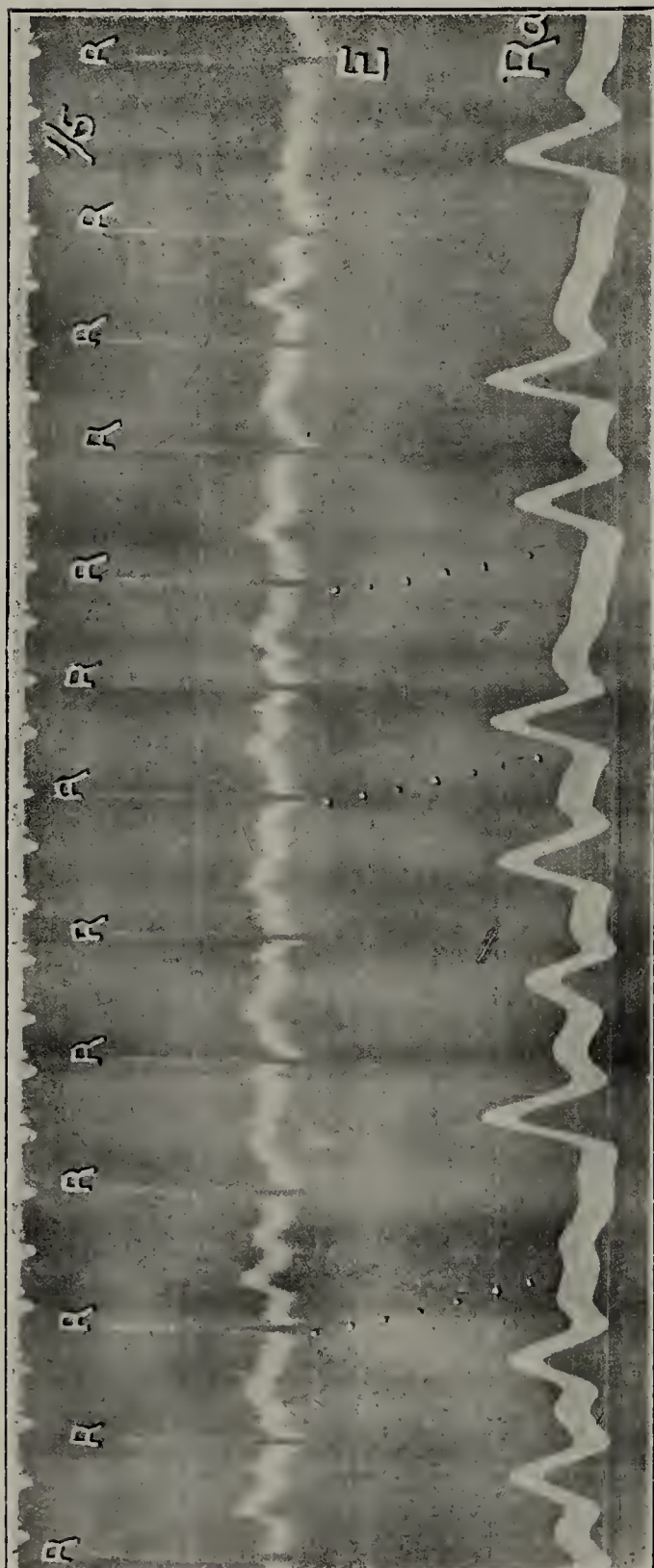


Fig. 154.

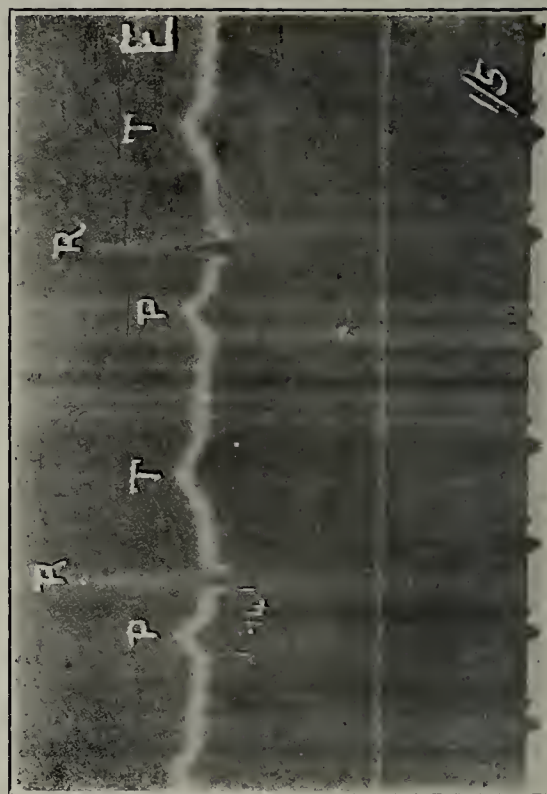


Fig. 155.

Fig. 154 and 155 (*Heart*, 1909-10, I, 306, *Fig. 15 and 16*). Electrocardiographic curves from a case of paroxysmal irregularity. Fig. 155 was taken two days after the subsidence of a paroxysm. Fig. 154 demonstrates the absence of regularity; the characteristic oscillations; the deformity of the variation *T*; the absence of relationship between height of peaks *R* and corresponding radial beats. There is no sign of a normal *P* variation. Fig. 155 shows the return of *P* and the shortening of the peaks *R*.

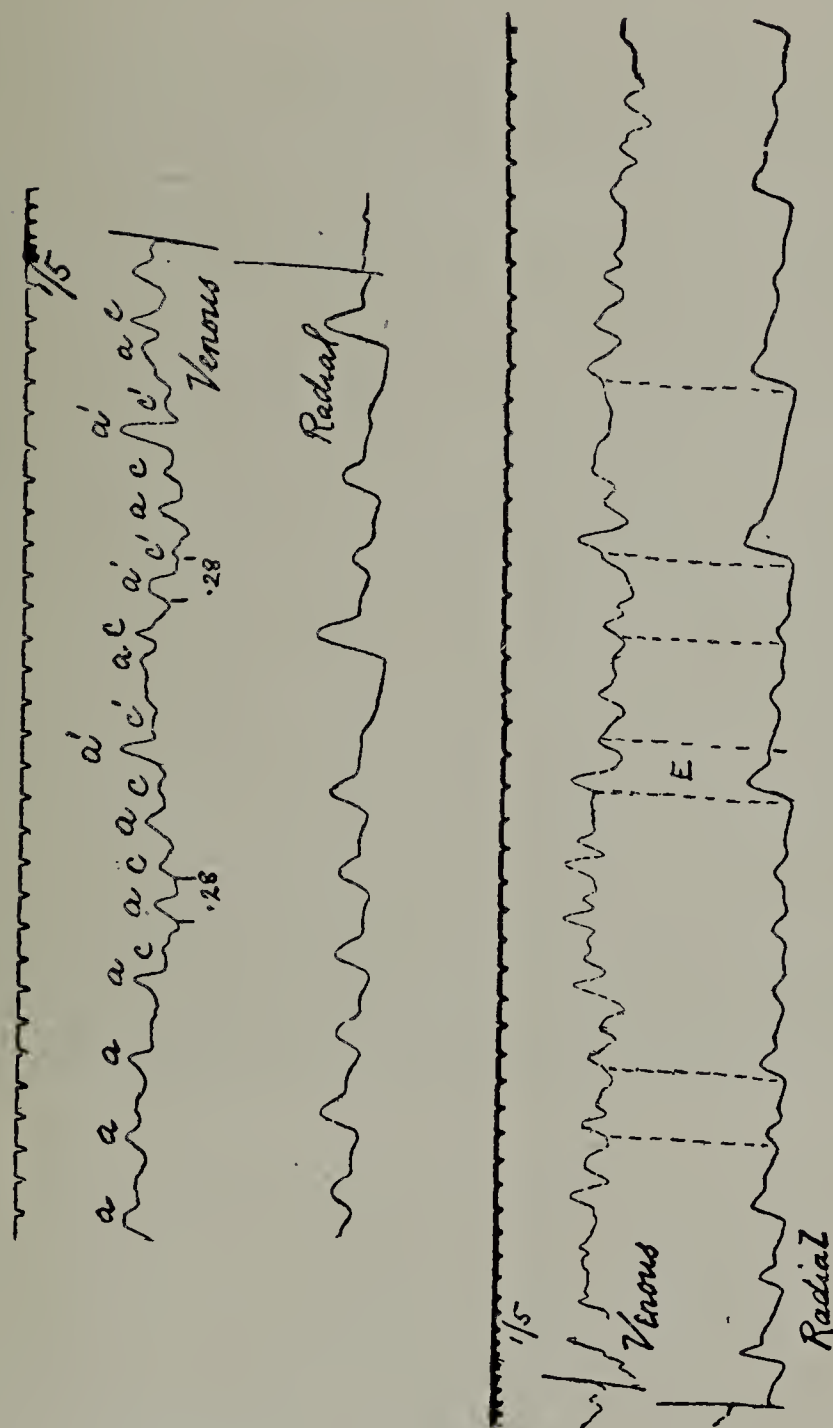


Fig. 156 A and B. (*Heart*, 1909-10, 1, 330). Two polygraph curves obtained from a case of mitral stenosis and taken within 12 days of each other. Tracing A shows a venous curve of the auricular form, and premature auricular contractions are present. The *a-c* intervals are increased. Tracing B shows a curve of the ventricular type, the pulse is completely irregular.

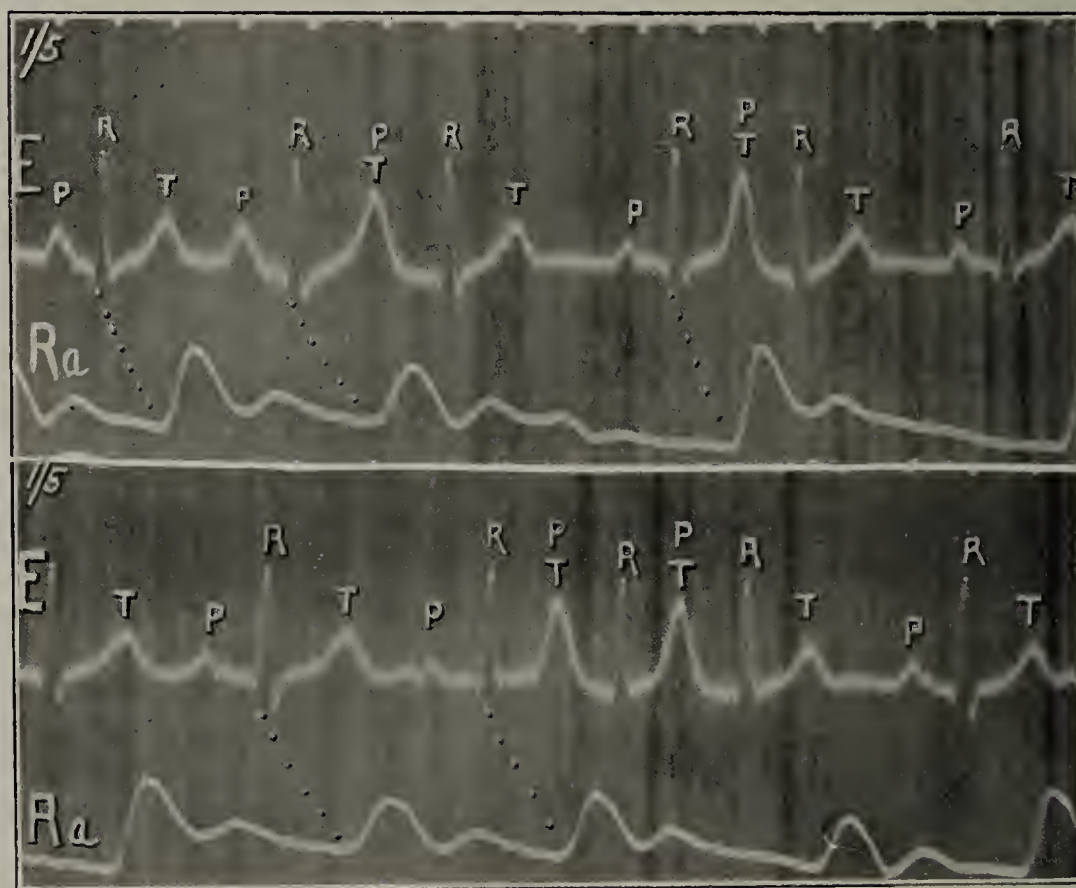


Fig. 157 ($\times \frac{2}{3}$) (*Heart*, 1909-10, I, 306, *Fig. 12*). Two photographic curves from a patient with mitral stenosis; taken before the onset of fibrillation. The electric curve and the radial tracing (*Ra*) show irregularities interrupting the sinus rhythm. They are premature auricular contractions. The *P-R* intervals are increased. In the second curve successive premature auricular contractions are shown.

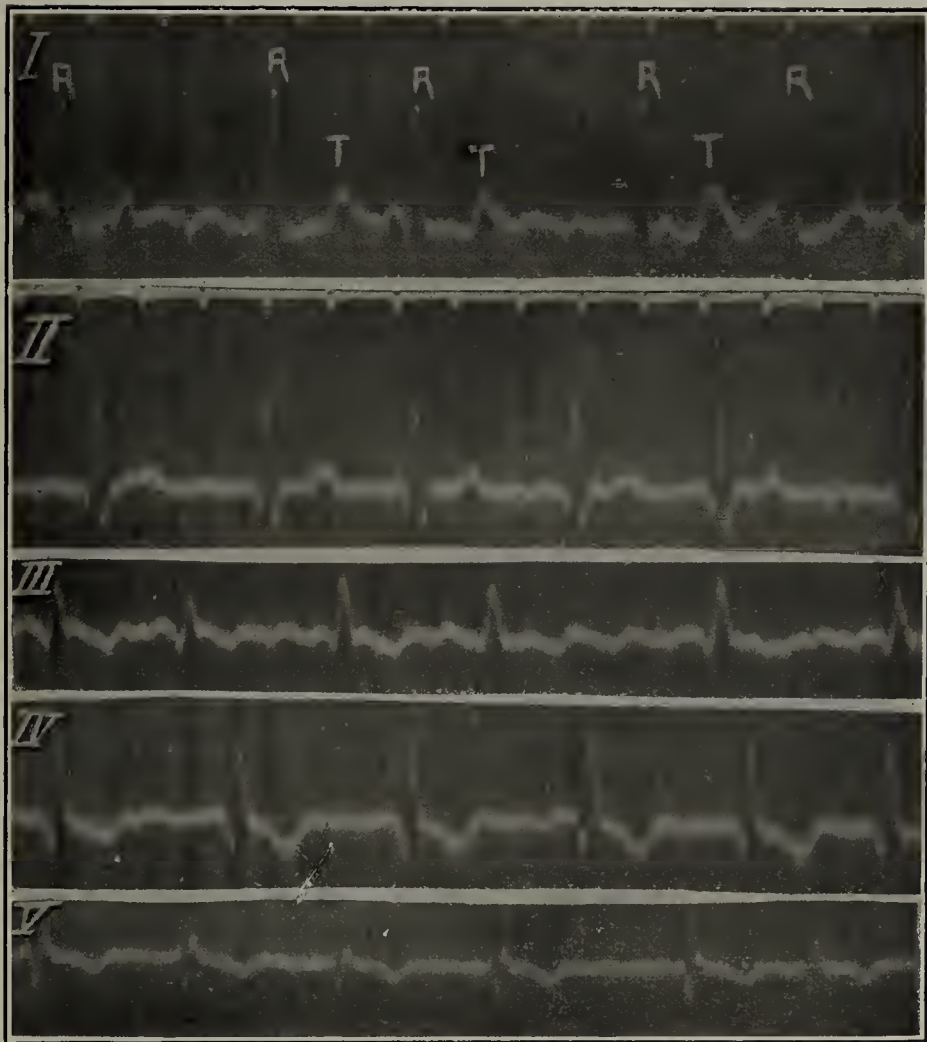


Fig. 158 ($\times \frac{2}{3}$) (*Heart*, 1909-10, I, 306, *Fig. 13*). From the same case, a few days after the onset of fibrillation.

- I. Lead from right arm and left leg.
- II. From sternum at junction of second cartilage and from apex.
- III. From sternum and point over right auricle.
- IV. From the third left space in anterior axillary line and from apex.
- V. From apex and abdomen, 3 inches internal to the apex and just below the ribs.

The first contact site mentioned above is in each case that which was connected to the lower end of the string.

Neither in Fig. 155 nor in Fig. 157, electrocardiograms taken from these two patients, are oscillations to be found, yet the curves were taken under conditions identical with those of Fig. 154 and 158. Such cases demonstrate conclusively the interdependence of the abnormal type of curve and the gross ventricular irregularity; and they afford a strong argument against the origin of the oscillations in the somatic musculature.

It is perfectly true that many electrocardiograms show traces of variations produced by contraction of the muscles of the body wall or limbs, and at times it may be difficult to exclude this complication from the curves. But the oscillations of which we are speaking bear no relationship to the extent of such movements. Muscular movements give rise to irregularities in the curves when a patient trembles or fidgets. In the great majority of such cases these extraneous vibrations can be identified at once by their general appearance and rate. If sufficient precautions are taken no such irregularities appear in subjects in whom the heart sequence is normal. Oscillations are invariably present in the class of patient considered, whatever the precautions adopted. They are of much the same degree from day to day and from hour to hour in the same subject. They are equally prominent when leads from the two arms are adopted, but vanish almost completely if the electrodes are attached to the two inferior extremities. The proposition, that they are part and parcel of the heart-beat as it is represented to us electrocardiographically, is unequivocal. Very numerous and special leads have been devised and employed for the exclusion of their origin in abdomen, limbs and head and neck. The special leads show that it is a matter of indifference, so far as the amplitude of the oscillations is concerned, as to how great is the extent of somatic musculature which lies beneath and between the electrodes. They demonstrate that the excursion is controlled by the proximity of the heart to the leads.

The special leads take us a step further, for they make it clear that it depends upon the part of the heart approached as to how conspicuously the oscillations appear.

The irregular oscillations arise in the vicinity of the auricle; the ventricular electric complex in complete irregularity is of the normal form.

The analysis, which the chest leads afford, is provided most strikingly by patients in whom the right auricle is enlarged, for in these a larger area of auricular muscle is in apposition to the chest wall; but the phenomena are not limited to such cases, though present in them in greater degree.

Fig 158 shows five separate leads. *I* is from the right arm and left leg. The remainder, *II-V*, are from the chest wall and were taken at the same time and under similar general conditions. *II* is the curve yielded by electrodes, one (the arm electrode) placed upon the sternum at the level of junction of the second rib, the other (or leg electrode) upon the apex.

The ventricular complex is represented by a curve similar to that often obtained in arm and leg leads from normal subjects; oscillations are also seen on the curve, but they are not prominent except towards its termination. *III* was obtained by moving the apex electrode to the fourth space on the right side and one inch from the sternal margin. The ventricular peaks are comparatively small, and the oscillations are maximal. The leg electrode was next replaced at the apex and the arm electrode was fixed in the third space in the anterior axillary line on the left side (*I V*); lastly the leg electrode was placed just below the costal margin three inches internal to the apex beat and the arm electrode at the apex beat (*V*). These two curves may be taken as representing in the main the pictures yielded by left and right ventricles respectively. They are almost entirely free of oscillations, and show regular and clean cut variations in the latter part of each systole.

The separate leads which we have examined, and numerous leads from other parts of the chest wall, demonstrate at the outset that the oscillations are conspicuous or the reverse according to the proximity or otherwise of electrodes to the right or superficial auricle. The conclusion is obvious and beyond question. Of the area of heart superficies, which is in relationship to the front of the chest wall, it is only over that portion of it which may be termed auricular that the maximal oscillations are produced, and as the heart is the source of the oscillations their origin may be traced to the auricular portion of it.

Separate leads from the chest wall have been obtained in twenty-five patients with the irregularity in question, and they all yield similar results. Two further examples are given in Fig. 159 and 182. The explanations of the figures may be consulted for the details of the leads.

The special curves give an analysis of those obtained by means of the usual arm-leg lead, and show that the latter are constructed by the superimposition of current curves of auricle and ventricle. They explain the deformation of the variation *T* and show that the variability of this summit from cycle to cycle is the result of the combination of the currents derived from the two sources, and that it is not an anomaly of the ventricular complex itself.

Those curves which are obtained by means of leads from the area overlying the auricle give the purest pictures of the oscillations which may be gained in the human subject. Obtained in this way they are found to vary in rate between 400 to 600 per minute, but the usual rate approaches 500 very closely.

Conclusions from the clinical findings.

The outstanding feature of the records from cases of complete irregularity of the heart is the entire absence of all sign of the normal auricular systole. It has been shown time and again that, while in normal subjects and in patients suffering from all other forms of heart irregularity or cardiac

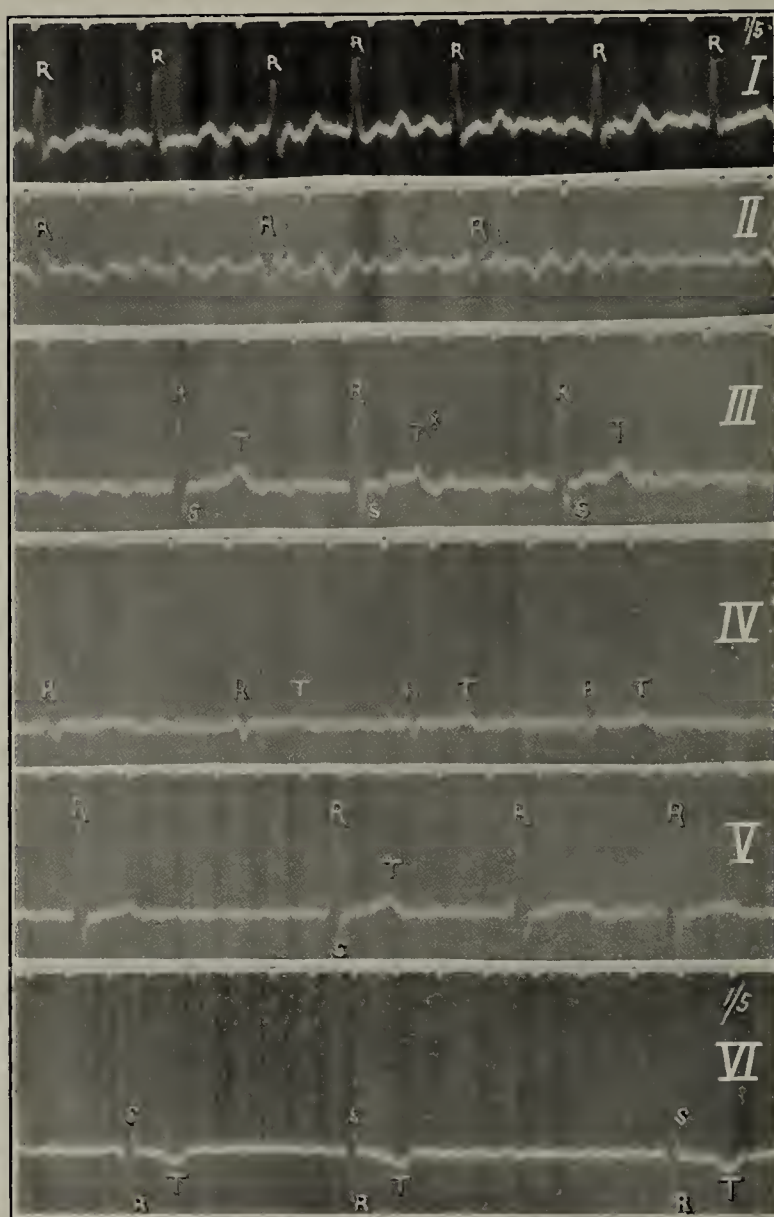


Fig. 159 ($\times \frac{1}{2}$) (*Heart*, 1909-10, 1, 306, *Fig. 14*). From a patient with mitral stenosis of rheumatic origin. Completely irregular heart action and the ventricular form of venous pulse were present. The figure shows electrocardiograms obtained by six separate leads.

- I. Right arm and left leg.
- II. Sternum between junction of second and third cartilages, and point over right auricle.
- III. Sternum and apex.
- IV. Sternum and outer end of first intercostal space.
- V. Outer end of third intercostal space and apex.
- VI. Apex and point on the abdomen, 3 inches internal to the apex and just below rib margin.

The first electrode site mentioned above is in each case the site of the electrode attached to the lower end of the string. The curves show that the oscillations are maximal according to the proximity of electrodes and superficial auricle. They also show the normal character of the ventricular complex.

disability the auricular systole leaves a definite impress either upon the cardiogram, upon the venous volume curve, upon the œsophageal curves^{16 & 36} or upon the electrocardiographic curve, such evidences of its normal activity are consistently wanting, whichever graphic method is employed, in the group of cases which is engaging our special attention.

The conclusion which it is impossible to avoid, is that the *normal presystolic* auricular contraction is in abeyance, temporarily or permanently.

Actual paralysis of the auricle has been suggested, but little support is now found for this hypothesis. The conditions of the circulation are often such that it is impossible to suppose that the pressure in the auricles is increased. The observation of hypertrophy in the auricle at autopsy led Mackenzie to abandon his earlier view of paralysis and influenced him in concluding that the auricle is active. The view was supported by the reappearance of signs of auricular contraction in paroxysmal cases. We are led to a precisely similar conclusion by the evidence yielded by the electrocardiographic curves. The auricle is the seat of an electric disturbance of a peculiar yet distinctive nature. The constancy of the oscillations, their unique appearance and *their presence throughout the whole of the cardiac cycle*, is responsible for the conviction that they are an essential feature of complete irregularity and that *the activity of the auricle is continual*.

Co-ordinate contraction of the auricle at any period of the cardiac cycle other than that of ventricular systole can be readily excluded. Co-ordinate and simultaneous contraction of auricle and ventricle can also be set aside, for, as we have seen, it gives rise to a totally different clinical picture.

In experimental work we encounter but one variety of auricular activity in which inco-ordination of the separate fibres is present, and this mechanism is one in which the auricle is in unceasing movement. It is the state known as fibrillation or delirium.

The final proof that complete irregularity of the heart in man is the result of fibrillation of the auricle, depends upon a close comparison of observations in man and animals. This comparison is given in the succeeding chapter.*

* A combined bibliography will be found at the end of the next chapter.

CHAPTER XVIII.

AURICULAR FIBRILLATION (*continued*).

Experimental and clinical electric curves compared.

ELECTRIC oscillations similar to those characterising complete irregularity of the heart in man are found experimentally when the auricle is fibrillating.

The oscillations of experimental auricular fibrillation were independently discovered by Rothberger and Winterberg³⁹ and by the writer.²⁰ Fibrillation of the auricle is readily obtained in experiment by faradisation of this chamber, and it usually continues for a lesser or greater time after the end of stimulation.

The oscillations which are obtained as a result of faradic stimulation of the auricles are unique, for they occur in no other experimental condition. They are seen in Fig. 160 and 163. They consist of variations which succeed each other rapidly, at a rate varying approximately from 500 to 900 per minute. They replace the usual *P* variations of the normal rhythm and produce the same deformation of the *T* variations as in the clinical curves.

Though never absolutely regular, yet at times the spacing is remarkable for its tendency to regularity, a quality noted in the clinical curves. They continue throughout the whole of the cardiac cycle.

The proposition that these variations are generated in the auricle and that they depend upon the delirium in the walls of this chamber is easily substantiated. They are only present when the auricle fibrillates, and are entirely absent from the curves yielded by the same animals when the normal rhythm is re-established. Fig. 160 and 161 are two electrocardiograms taken from a single animal within a few minutes of each other. The first, during an auricular fibrillation period, exhibits the oscillations; the second, after the re-establishment of the normal sequence, shows none, but the *P* variations have reappeared.

Their dependence upon fibrillation is also suggested by the following considerations.

It is well known that auricular fibrillation may be of many grades, the movement of muscle levers is fine or coarse in the same experiment or from animal to animal. It is only rarely that an actual correspondence can be found between the individual muscle movements and the electric variations, and this is to be expected, for the whole auricle is active, and

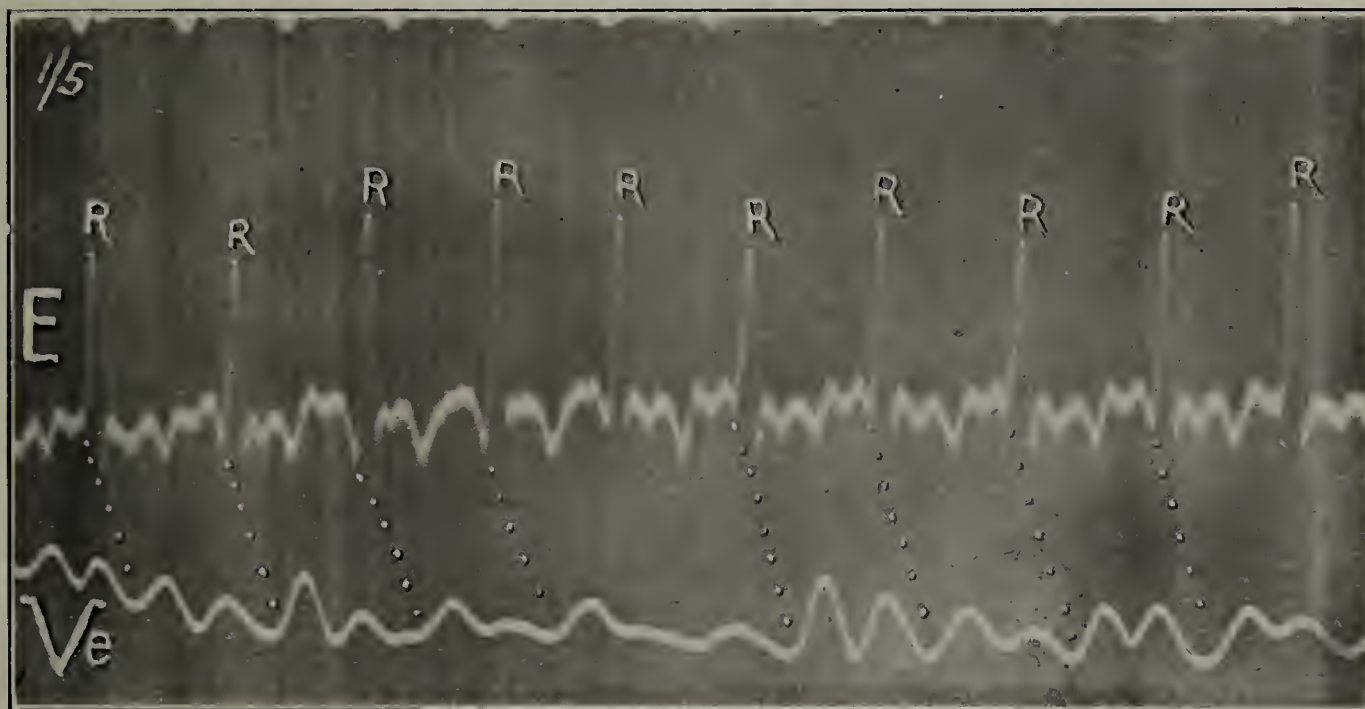


Fig. 160 (*Heart*, 1909-10, 1, 306, *Fig. 20*). Experimental curve. Taken from a dog with chest wall intact, while the auricle was fibrillating. Venous and electrocardiographic curves are shown. The venous curve is ventricular in form; the electrocardiogram shows irregularity of the ventricle, and well marked auricular oscillations.

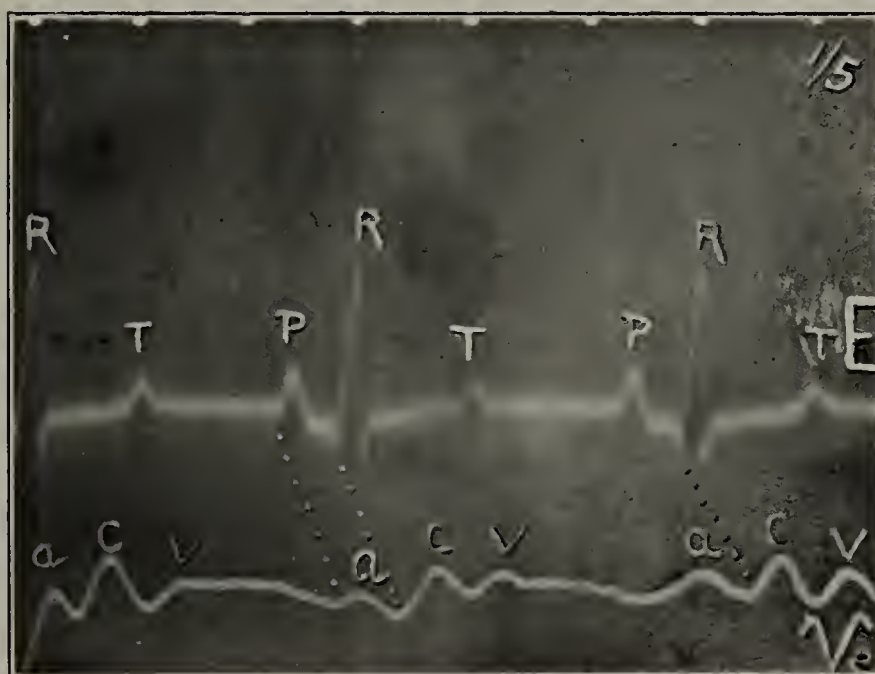


Fig. 161 (*ibid*, *Fig. 21*). From the same animal a few minutes later. The venous pulse is now auricular in form: the electrocardiogram shows the return of *P*, while the oscillations have vanished with the fibrillation.

only a small portion of the muscular movement is recorded. Nevertheless, correspondence may be found in some degree. When the muscular movement is small and the vibration of the lever is rapid, the oscillations tend to be of small excursion and, as seen under vagal stimulation, may reach a rate of 900 or more per minute.* With the coarse fibrillary movements on the other hand, the electric variations are of greater amplitude and are slower. It would seem that the oscillation appearing in auricular fibrillation is the total result of the electric changes occurring in the individual fibres, and that similarly the movement of the myocardiograph lever is the total result of the combination of contractions and relaxations of individual fibres. If at any given time a greater number of fibres are in contraction than in relaxation, and such a state would be anticipated when the auricle is tending to return to a normal or co-ordinate mechanism, then a tendency to general contraction of the chamber as a whole, and an inclination for the electric variations of given direction to superimpose and produce a more or less prominent resultant curve would be present.

The location of the electric change in the auricle is a simple matter if direct leads are taken from the heart itself, and from various parts of the body wall. The oscillations are maximal when the lead is from the auricular substance (Fig. 162 *I* and *II*), and their amplitude decreases according as the distance separating auricle and electrode is widened (Fig. 162 *V* and *VI*). Curves taken directly from the ventricle show comparatively little trace of the oscillations while the auricle is fibrillating (Fig. 162 *III*).

There is therefore no question but that, in experimental auricular fibrillation, oscillations in the electric curve are produced which may be definitely located as arising in the auricle; there is equally no doubt that the oscillations are produced as a result of the fibrillation, for they are contemporaneous with it and with no other mechanism.



The electrocardiographic curves compared in more detail.

The oscillations constitute one of the outstanding features in the electric curves of both the clinical condition and experimental fibrillation. These oscillations vary considerably in form, in rate, and in extent from case to case, and from experiment to experiment. But when the material for selection is abundant it is often possible to choose examples of curves which are alike pictorially. For purposes of pictorial comparison two curves have been selected, and are shown in Fig. 163 and 164. The first is an experimental curve, and the second is from a case of mitral stenosis with completely irregular pulse. The similarity is striking. In instances where the ventricular

* Speaking of the more prominent oscillations only.

beat is rapid (Fig. 154), and where on this account the oscillations are obscured, the nature of the rhythm is identified not so much by searching for oscillations as by noticing the deformity of those portions of the curve which lie between the adjacent peaks *R*, *R*. The pieces of curve referred to show no resemblance to each other. A similar example but an experimental one is shown in Fig. 185 (the last half of the curve). The first half of this curve represents the escape following vagal stimulation. The oscillations, which are so completely unmasked by the prolongation of diastole, escape attention in the succeeding cycles.

Again, there is the fact that in experimental as well as in clinical curves the general character of the ventricular complex is unaltered. In the dog this can be readily demonstrated by leading from any two points of the ventricular surface. The same type of curve is yielded whether the auricle is fibrillating or in co-ordinate contraction. Fig. 162 *III* and *IV* may be compared. The former, taken while the auricle is fibrillating, shows a faster heart beat to the right and the last phases of the shorter cycles are curtailed. To the left of the same figure, the full complex is shown while the heart is escaping from the inhibitory slowing. The ventricular complexes are of the same form as those exhibited while the sequence is normal, and the heart regular (Fig. 162 *IV*). The same fact may be shown when the oscillations lack prominence; leads from the upper and lower part of the chest of the same animal may give similar and normal ventricular curves with one or other mechanism present (Fig. 162 *V* and *VI*).

The electrocardiographic curves, experimental and clinical, are alike in every other respect. The irregular distribution of the ventricular peaks *R*, the direction of these peaks (direction of base negativity) and the submerged variation *T*, are features held in common. There is a further characteristic, which deserves more attention. It is common to both clinical and experimental curves. There is no fixed relationship between the heights of the peaks *R*, and either the pauses which precede them or the height of corresponding carotid beats. The absence of both relationships is shown in Fig. 154 and 185. The same disproportions are seen in the condition known as heart alternation (Chapter XXIII), and it is not improbable that a common factor aids in its production under the two sets of circumstances. But though a phenomenon of this sort plays some part, yet in certain of the records it is obviously a minor factor. It will be clear that when the oscillations are extensive in amplitude that much will depend upon the relative positions of the peak *R* and the summit of an auricular oscillation. The individual oscillation may be regarded as  shaped; if a peak *R* falls with the depression between two adjacent oscillations, that peak will be relatively short; if it falls on the summit its height will be enhanced; while falling on the side of the  an intermediate amplitude may be expected. The oscillations in Fig. 163 and 164 are not spaced with absolute regularity, but it is possible to reconstruct those oscillations which are distorted by *R* variations coinciding with them.

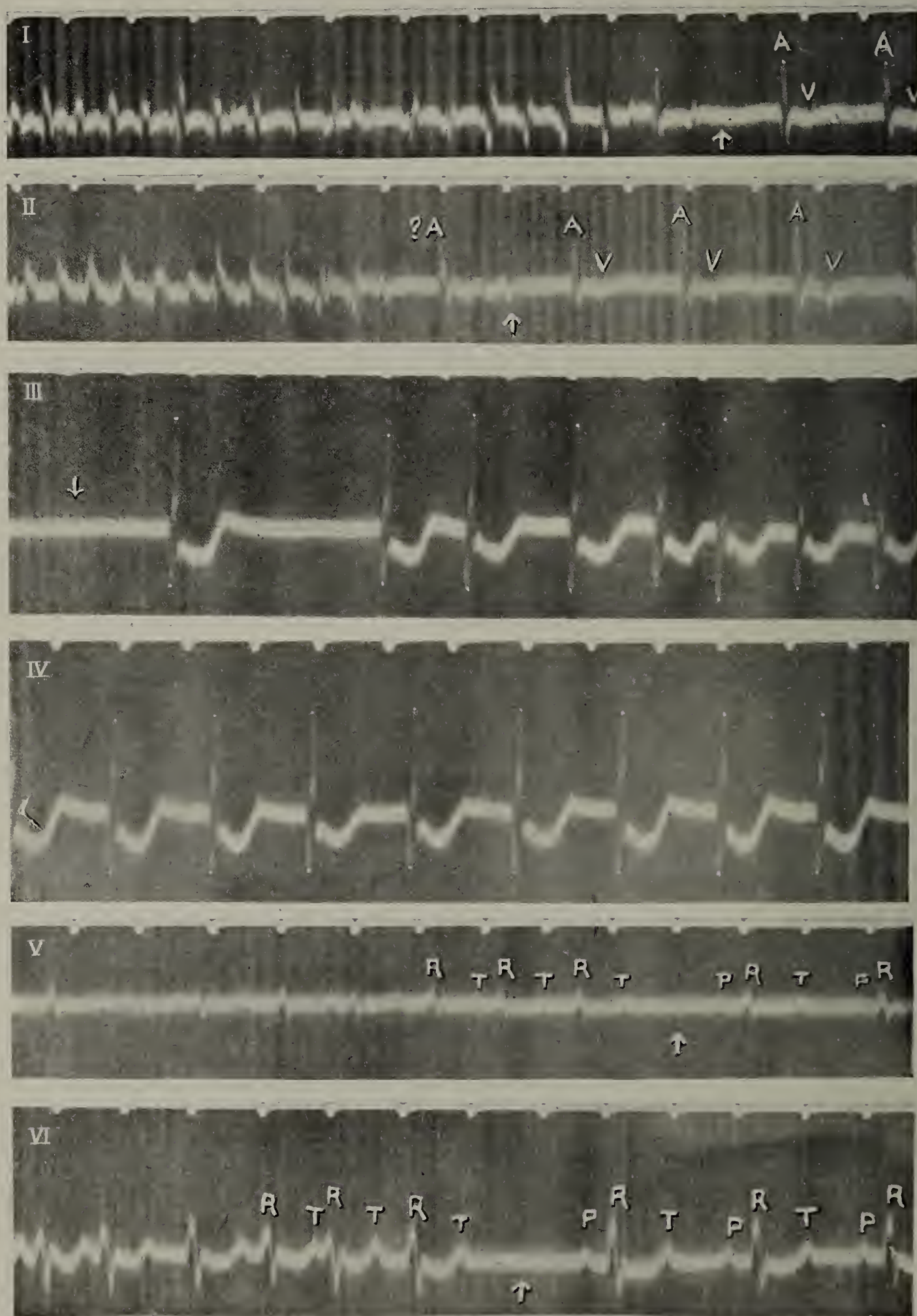


Fig. 162 (*Heart*, 1909-10, I, 306, *Fig. 31*). A series of experimental curves from a single animal.

- I. Lead from a point on the auricle just below the superior vena cava and from the inferior vena cava. As a result of faradisation the auricle was fibrillating up to the point marked by an arrow. At this point the co-ordinate contractions returned. A =auricular variation. V =ventricular variation.
- II. A similar curve. The point where auricular fibrillation ceased is uncertain. In both curves the oscillations are maximal.
- III. Lead from two points on the right ventricle, one above and near the right border, and one below and near the apex. The auricle was in fibrillation over the stretch of the whole curve, yet no oscillations are seen. Up to the point where the arrow is placed the vagus was stimulated and a slight escape of current is shown in the opening phases of the curve.
- IV. Lead from the same points after the return to the normal rhythm. A comparison with the last curve shows that the direction of contraction in the ventricle is identical while auricular fibrillation and auricular co-ordination are present.

Curves *III* and *IV* may be compared in respect of the amplitude of the opening variations of the separate beats. In *III* it is variable, in *IV* it is constant. The auricular oscillations are not entirely responsible for the variation in the height of the peaks R during fibrillation, for in *III* no oscillations are present. The amplitude of the opening variations is greater in *III* than in *IV*.

- V. Lead from the upper and lower ends of the wound. The wound travelled through the centre and whole length of the sternum. The upper electrode was upon the base of the neck, the lower one upon the diaphragm. The curve is very small and though much somatic musculature was included between the electrodes, no oscillations are seen. At the arrow the normal rhythm was resumed. Up to this point the sensitivity of the galvanometer was maintained at a constant pitch.
- VI. The same lead as the last, but the sensitivity of the galvanometer was increased approximately threefold. The oscillations are just visible and distort the curve to some extent. The normal rhythm was resumed where the arrow is placed. A careful comparison of the ventricular curves before and after the resumption of the normal rhythm shows them to be of essentially the same form, and this form is the normal one. A comparison of the five curves shows that the oscillations arise in the auricle.

The electrode site first mentioned is invariably the site of the electrode attached to the lower end of the string.



Fig. 163 and 164 (*Heart*, 1909-10, I, 306, *Fig. 22 and 23*). Two electrocardiograms, the upper from a dog in which the auricle was fibrillating, the second from a case of mitral stenosis exhibiting complete irregularity and the ventricular form of venous pulse. The two curves are almost identical. Each shows the characteristic oscillations, the irregularly placed *R* peaks which vary in height according to their relationship to oscillations, and the absence of true *P* variations.

It is found that where a peak *R* falls at a point at which the summit of an oscillation is anticipated the peak is tall in comparison to one which falls where a depression is expected. On the other hand there are many curves in which this explanation is insufficient. Fig. 162 *III*, is an experimental curve taken by means of a direct lead from the right ventricle during fibrillation. Oscillations are entirely absent, yet there is no relationship between the amplitude of preliminary variations and the length of preceding pause.

Reviewing the electrical phenomena as a whole it becomes manifest that the clinical and experimental curves resemble each other in every detail. The close pictorial resemblance in many instances, and the absolute identity of all the essential characteristics taken alone or in relation to corresponding events, are in themselves sufficient to carry conviction of a similar mechanism in clinical and experimental instances.

The arterial curves compared.

In 1906, Cushny and Edmunds ^{3 & 4} investigated a case of paroxysmal irregularity of the heart. Unfortunately no venous curves were taken, but from the radial tracings it is in the highest degree probable that the irregular heart action with which they were dealing was of the nature here considered. Attention was drawn in this paper to the close similarity of the radial curves taken from their patient and arterial curves obtained from dogs in which the auricle was in a state of fibrillation. They remarked



Fig. 165 ($\times \frac{2}{3}$) (*Heart*, 1909-10, 1, 340). Hürthle manometer curves from the carotid of a dog, in which the auricle was fibrillating. Chest wall intact. A small portion of normal curve is shown in line 3. At the point where the arrow is placed the auricle, which had resumed its normal rhythm, was faradised and fibrillation was re-established.

in particular upon the absence of relationship between the height reached by the arterial beats and the pauses preceding them.

In so far as the experimental arterial curves are concerned, little can be added to the description given by Cushny and Edmunds. The irregularity of the arterial pulse in auricular fibrillation is absolute and has the same qualities as those presented by the curves in complete irregularity of the heart in man. The rate of the ventricles is increased. Some examples of the Hürthle manometer curves taken from the carotid of a single animal are given in Fig. 165. A short strip of normal curve, the only piece in the figure, is shown in the third line. At the point where the arrow is placed, the auricle, which had spontaneously ceased to fibrillate, was faradised once more and the fibrillation was re-induced. The curves were taken with the chest wall intact and may be compared with the radial tracings given in the preceding chapter (Fig. 149, etc.).

The venous curves and their comparison.

The venous curves associated with complete irregularity of the heart have been discussed at length in the last chapter.

In Fig. 166 and 167 strips of curve from a single animal are represented. In Fig. 167 the normal rhythm is interrupted by a short induced paroxysm of irregularity due to fibrillation of the auricle. The venous tracing while the rhythm is regular, before and after the paroxysm, consists of clearly inscribed *a*, *c* and *v* waves. During the paroxysmal period each of the irregular beats (several fail to impress the femoral curve) is accompanied by two prominent waves in the veins; the waves are separated from each other, and the second wave is succeeded by a well-marked depression. The main waves fall in the systolic interval, *E*, but where the pause of diastole is long a stasis wave is also apparent. Fig. 166 shows the onset of a paroxysm, and the venous curve at its commencement is of the usual type. Towards the termination of the curve there is a succession of more rapid beats and the corresponding venous curves incline to the plateau form; the systolic depression of the slower beats is wanting. At no point in either tracing are true presystolic waves discoverable while the auricle is fibrillating. The venous records are of precisely the same type as those which characterise the clinical condition. The figures may be compared most profitably with Fig. 150 *A* and *B*. The variation in form with the enhanced rate of ventricular beats has its clinical parallel. Further variations are met with which seem to indicate that the plateau type of venous curve may result from cardiac dilatation. The plateau form is more commonly found towards the end of an experiment. A third curve, in which the rate of the ventricle is slow and which was taken towards the termination of an experiment is shown in Fig. 168. The beats in the venous curve are of the characteristic

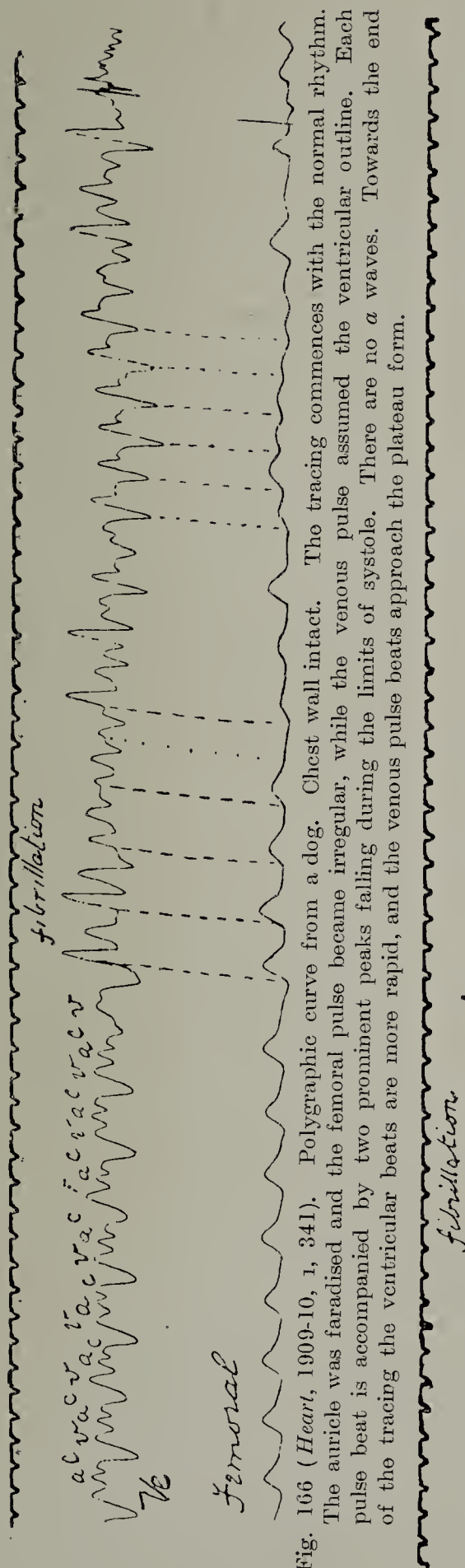


Fig. 166 (*Heart*, 1909-10, 1, 341). Polygraphic curve from a dog. Chest wall intact. The tracing commences with the normal rhythm. The auricle was faradised and the femoral pulse became irregular, while the venous pulse assumed the ventricular outline. Each pulse beat is accompanied by two prominent peaks falling during the limits of systole. There are no α waves. Towards the end of the tracing the ventricular beats are more rapid, and the venous pulse beats approach the plateau form.

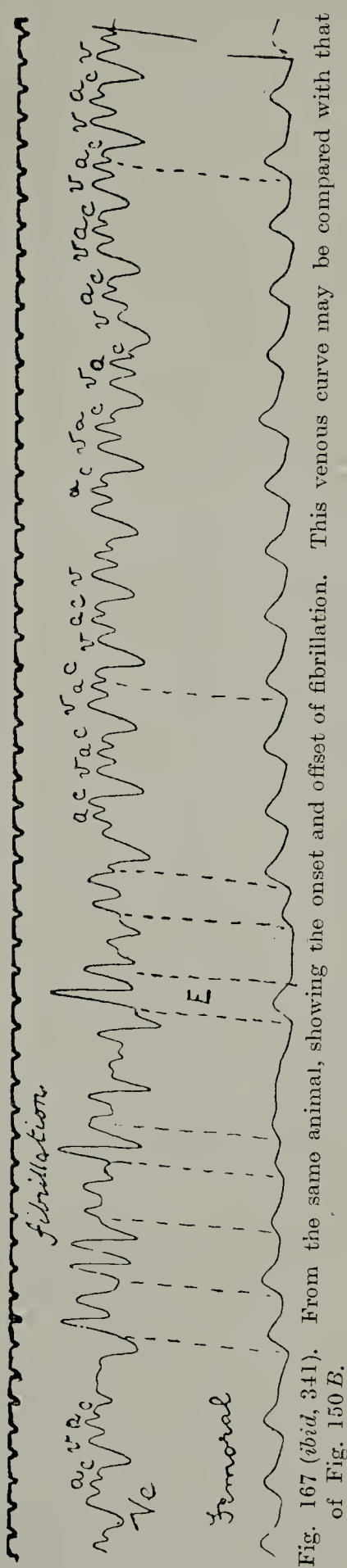


Fig. 167 (*ibid.*, 341). From the same animal, showing the onset and offset of fibrillation. This venous curve may be compared with that of Fig. 150 B.

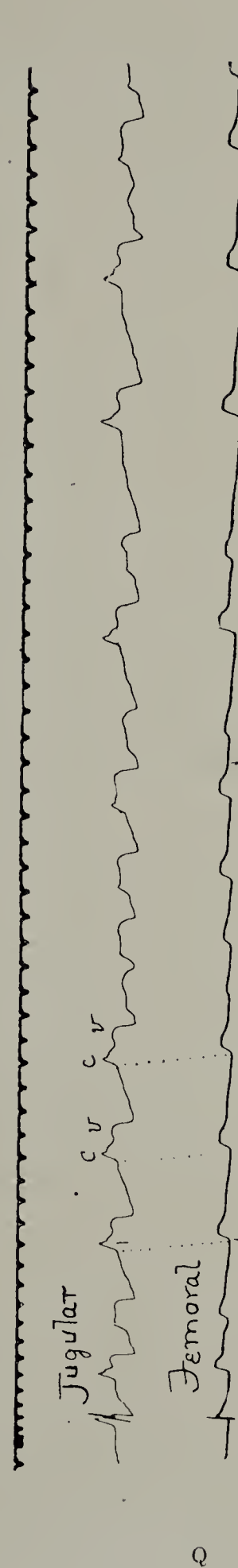


Fig. 168. Polygraphic curve from a dog in which the auricle was fibrillating and in which the ventricle was beating slowly and regularly in response to it. This type of venous pulse is very characteristic; it may be compared with Fig. 150 A.

plateau form ; (the curve is very similar to those which have been recently published by Rihl.³⁸)

When the ventricle beats slowly, it is not uncommon to find small and rapid oscillations in the diastolic portions of the venous curves of patients. The appearance of these small undulations of pressure in the veins in the clinical condition finds its parallel in experiment. The waves are not found in all cases, experimental or clinical ; a slow ventricular action is essential (cp. Fig. 152 A, and the discussion relating to it). They are found in experiment when the ventricular rate is retarded by stimulation of the vagus. They are well seen in the long diastoles of Fig. 169. The slowing of the ventricle in this experiment was induced by excitation of the vagus. The auricle was in fibrillation throughout and the little waves are the result of its activity. They are also shown in Fig. 186. While their dependence upon fibrillation in the experimental case cannot be gainsaid, a similar origin is not demonstrated with equal facility in the clinical instance. At the same time an additional observation is helpful. In man the rate of the venous waves is approximately the same as the rate of the electric oscillations. Simultaneous electric and venous curves are shown in Fig. 170. The oscillations are clear in both lines, and the rate is approximately 450 in each.

Reviewing the observations upon the venous pulse in complete irregularity of the heart and in experimental auricular fibrillation we may state that the records are alike in every respect.

A summary of the comparison instituted between the records obtained in complete irregularity of the heart in man and in experimental auricular fibrillation.

The comparison between complete irregularity of the heart in man, and auricular fibrillation in the dog is now complete. It has been demonstrated that the clinical and experimental conditions resemble each other in every respect in which they have been investigated. The observations are summarised in the following table, which consists of a systematic list of the features presented in common.

The radial curves.

1. *Rate increased as compared with the normal.*
2. *Presence of absolute irregularity.*
3. *Absence of fixed relationship between the strength of a beat and the preceding pause.*
4. *The failure of many beats to reach the arteries, and the presence of beats of all shapes and sizes.*

The venous curves.

1. *The presence of the ventricular form of venous pulse (the absence of "a" waves).*

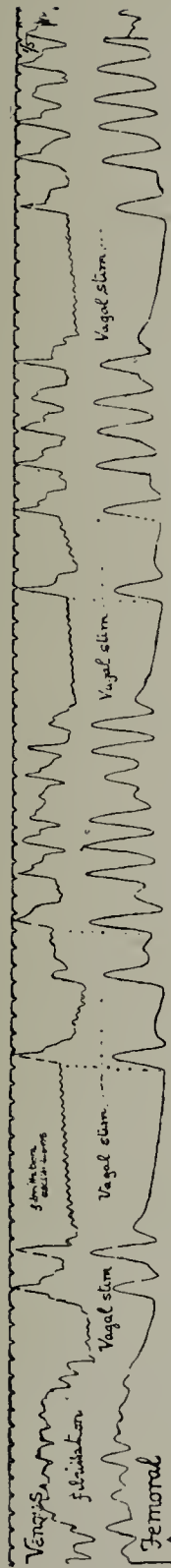


Fig. 169 ($\times \frac{1}{2}$) (*Brit. Med. Journ.*, 1910, II, 1670). Venous and femoral curves from a dog. The auricle is fibrillating throughout, and the venous pulse is of the ventricular form. When the vagus is stimulated the ventricle ceases to beat, but the auricle continues to fibrillate, and leaves its impress on the venous curve in the form of small and rapid oscillations.



Fig. 170 ($\times \frac{2}{3}$) (*Heart*, 1909-10, I, 306, Fig. 19). Simultaneous electrocardiogram and venous pulse curve from a case of auricular fibrillation in which the pulse was slow. Oscillations occur in both curves, and they are of approximately the same rate in each.

2. *The definite variations in the form of the individual venous pulse beats under certain conditions.*
3. *The presence during diastole of rapid undulations of venous pressure when the heart beat is slow.*

The electrocardiograms.

1. *The occurrence of a base negative variation " R " at the commencement of ventricular systole.*
2. *The presence of a variation " T " which is deformed in leads from the extremities, but which is clear cut and of normal form in leads from the heart itself.*
3. *The absence of an auricular systolic variation " P."*
4. *The occurrence of oscillations, generated in, or in the vicinity of, the auricle. Their persistence throughout the whole cardiac cycle. The rapidity of the oscillations. Their disappearance when the normal rhythm is resumed.*
5. *The absence of fixed relationship between the height of the peak " R " and the length of the preceding ventricular pause. The absence of relationship between the height of " R " and the height of the corresponding primary waves of the arterial pulse.*

Auricular fibrillation as a clinical entity.

The clinical and experimental comparison employed as a deliberate test of the proposition, originally based upon clinical facts, is invaluable. Without it our attitude towards a large group of clinical cases would remain in the hypothetical stage. With the comparison hesitancy gives place to a feeling of security ; we may approach our patients with a true appreciation of the affection which we so frequently encounter ; and the meaning of many otherwise obscure manifestations becomes clear.

Where, in conjunction with an absolutely irregular pulse, the ventricular form of venous pulse and the characteristic electrocardiogram are present, the evidence is complete. But, as in a large number of patients no case exhibiting complete irregularity and the ventricular form of venous pulse has been found, which does not show likewise the typical electric records the electrocardiograms cannot be held as essential to the diagnosis. Moreover, as no case of complete irregularity has been recorded in which the venous pulse may not be interpreted as of the ventricular form, the conclusion may be carried a stage further. There is no hesitation in stating that in the vast majority of cases of complete irregularity of the heart, auricular fibrillation is responsible for the disturbance of ventricular rhythm. It is sufficient if a single strip of curve is obtained from the radial artery, and if no two beats on the strip are of the same length

(and given, of course, that the heart rate is not manifesting a gradual acceleration or retardation while such a strip of curve is recorded) for the statement to be made that auricular fibrillation is present in all but extremely exceptional cases.* It may indeed be affirmed that auricular fibrillation with its complete irregularity accounts for approximately 50% of all persistent irregularities of the heart. And in dealing with cases of advanced mitral stenosis admitted to hospital it may be said that the presence of auricular fibrillation is the rule.

The possibility of the occurrence of auricular fibrillation as a clinical phenomenon has been recognised by several writers. It was suggested by Cushny.² It was also mentioned by Mackenzie,²⁹ and Wenckebach⁴⁶ discussed it briefly. But the proposition received no real measure of support—it had been suggested only in rare and isolated cases—until the electric and venous curves obtaining in the experimental and clinical conditions were studied in detail and side by side (Rothberger and Winterberg and the writer). These observations have placed the whole question upon a secure basis, and the presence of auricular fibrillation in clinical cases has now met with wide acceptance. There remains but a single argument in opposition to the view.

It has been said that it is inconceivable that the heart should remain in this state for long periods. Janowski¹⁵ recorded a case of complete irregularity of $5\frac{1}{2}$ years' duration, and Mackenzie has watched similar patients for even double this time. In reply, the persistence of tremor in the tongue following nerve section and the well-recognised and chronic fibrillary twitching of the voluntary musculature in many nervous maladies have been instanced. Fibrillation of the ventricle, it is true, is accompanied by almost instant death, for fibrillation in this chamber brings the circulation to a prompt standstill. The auricles are not indispensable to the circuit of blood in the body, but may be regarded rather as temporary reservoirs which accommodate the blood flowing to the heart while the ventricle is in systole. The large veins are perfectly capable of subserving this function, and the resulting general disturbance of the circulation is but slight. We have perfectly definite evidence that the normal auricular contraction is in abeyance in a large number of patients in whom the peripheral circulation is unhindered, and auricular fibrillation may last for many hours in a dog and yet the blood-flow in the body is fair throughout. Therefore, on the ground that auricular fibrillation is incompatible with a continued circulation, this argument may be entirely dismissed. If it is implied that the hindrance would occur, not in the auricle, but in the ventricle in virtue of the high grade of the disorder of its rhythm, it may be replied that such an argument is but a denial of an ascertained fact. The secret of the lack of disturbance as a result of the ventricular irregularity is found in the direction taken by the wave of contraction in the ventricular musculature.

*The solitary exceptions are cases of gross sinus arrhythmia, (Fig. 200).

The electric records testify that the contraction route followed is normal. Strong and weak contractions may be mixed together in profusion, but all the beats will tend to be effective in expelling the ventricular contents, and the majority of the beats are effective.

Moreover, the *a priori* arguments are completely overthrown by the following observations, which in themselves fully establish the relationship of auricular fibrillation with complete irregularity of the heart.

Auricular fibrillation in the horse.

As a rare affection complete irregularity of the heart occurs in horses.^{22 & 24} It is accompanied by marked dyspnoea, easily provoked exhaustion, marked systolic venous pulsation on slight exertion, and sometimes by dropsy and epistaxis. The heart rate is increased (from the normal rate of 35-40 up to 60-70 or even to as high a rate as 150 per minute). The disordered action is of characteristic form; the pauses show the usual and typical lack of uniformity. Of five horses with heart irregularity, which have been examined, four presented this type of arrhythmia; the fifth was an instance of single premature contractions. Observations have been made upon several animals. Curves have been obtained from artery and vein, but not synchronously, the timing of the jugular upstroke has therefore been subjective, but it is reliable where the pulse is slow (50-70). The curves are of the usual types. On one occasion electrocardiograms have been obtained. Examples of them are shown in Fig. 171. The absolute nature of the arrhythmia is apparent; as also is the supraventricular origin of all the impulses, and the absence of *P* variations. A faint trace of the oscillations is apparent from place to place in the curves. This animal was shot and the chest opened under artificial respiration. Unfortunately the right ventricle was damaged in the process; the auricle however was seen to be in a state of fibrillation. The damage to the ventricle during the progress of this observation necessitated its repetition, and an opportunity has recently arisen through the kindness of Colonel Blenkinsop of Salisbury. A horse presenting the characteristic symptomatology and exhibiting a gross disorder of the ventricular rhythm (pulse rate 50-70) was shot through the brain, the chest was opened in a similar manner, and the heart watched for five minutes or more. The ventricular movements were of the most disorderly nature. The auricle stood in the diastolic position, and close inspection of it revealed the presence of fine inco-ordinate movements, local twitchings and vermicular contractions. The appearance was characteristic.

The investigations of these animals should disperse the last remaining doubt of the existence of auricular fibrillation as an important pathological condition.

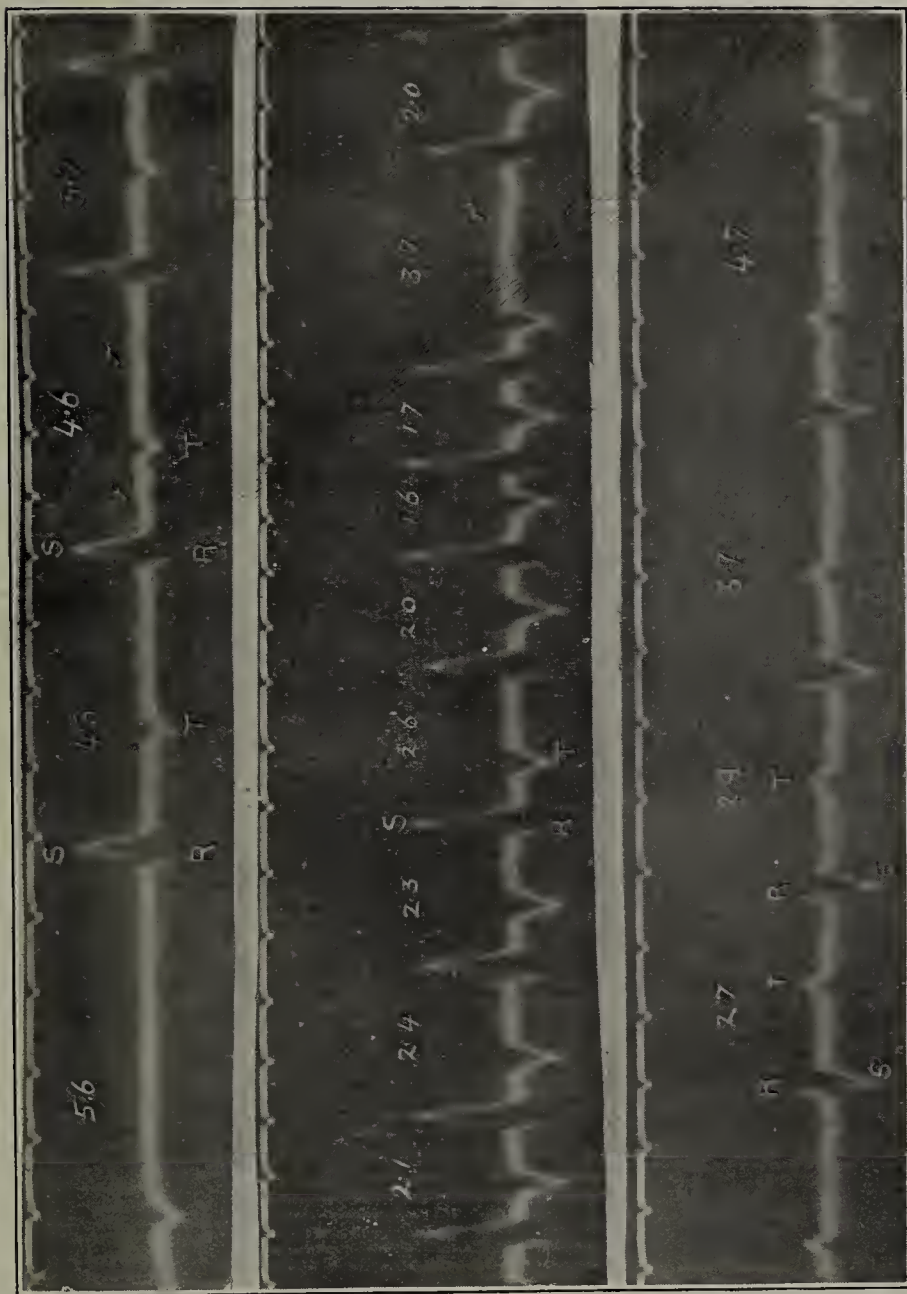


Fig. 171. ($\times \frac{5}{8}$). Three electrocardiograms from a horse, the third strip is from the right fore-limb and left hind-limb. Complete irregularity of the heart is present, each heart cycle is represented by *R*, a large *S* and a *T* variation; there is no sign of *P*. There are traces of oscillations in the longest diastolic periods shown.

The first two strips were taken from the epigastrium and centre of the breast in front; (they are consequently inverted). They show the same characters as those exhibited by the third strip. The irregularity is of high grade, the heart rate at times exceeds 150 per minute (normal 35-40). Oscillations are most distinct in the first strip. *P* is completely absent. From a horse in which the auricle was seen to be in fibrillation.

The morbid anatomy in clinical auricular fibrillation.

The changes found in the heart in clinical fibrillation have been reported by a number of workers,^{7, 18, 30, 42 & 43} but the study is by no means complete at the present time. The heart musculature as a whole shows a more or less marked grade of chronic inflammatory change, proceeding to fibrosis, which falls with particular severity upon the auricular tissues, especially at the inlet of the superior vena cava. The diseased condition may be well marked in the neighbourhood of the inferior vena cava and coronary sinus; similar changes have been described in the auriculo-ventricular bundle, and in scattered areas of the ventricular musculature. Special stress is laid by most writers upon the condition of the sino-auricular node and the musculature uniting the superficial aspect of superior vena cava and right auricle. The fibrosis is finely divided and diffuse and is usually accompanied by lymphocytic infiltration and increase of connective tissues and its cells, occasionally by leucocytic deposits of the polymorphous variety. Where these collections are intense, the muscle fibres are atrophied and separated. Larger areas of denser fibrosis are not uncommon and are found in both auricle and ventricle.

At the present time it is impossible definitely to correlate the morbid changes with the mechanism as we know it to exist; for changes of equal intensity and of similar nature are met with in association with a normal heart rhythm.³⁴

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CHAPTER XIX.

THE ASSOCIATION OF TACHYCARDIA OF AURICULAR ORIGIN AND AURICULAR FIBRILLATION WITH HEART-BLOCK. THE ACTION OF DIGITALIS.

Acceleration of the auricle and heart-block.

In the chapter which deals with clinical heart-block, we saw that, where partial heart-block is present, the ratio of the auricular to the ventricular rates is controlled by two factors, the auricular rate, and the functional efficiency of the junctional tissues. Under normal conditions the junctional tissues are capable of transmitting impulses at a much faster rate than is required of them. Furthermore, with acceleration of the sinus rate, it is not uncommonly found, both experimentally and clinically, that the *As-Vs* interval actually decreases when this acceleration is marked.¹³ An increase in the capacity of conduction seems to accompany a general call upon the heart for more work.

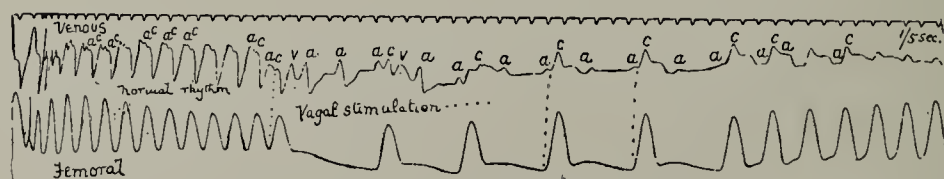


Fig. 172. ($\times \frac{1}{2}$) (*British Medical Journal*, 1910, II. 1670). Venous and femoral curves from a dog showing the effect of vagus stimulation. With the stimulation 2 : 1 heart-block appears.

The phenomena stands in contrast to the response of the damaged heart. The reaction of the heart under these circumstances introduces us more closely to the subject of the factors influencing one of the prime determinants of auriculo-ventricular ratio, namely, the functional capacity of the tissues uniting the two chambers. The functional capacity is modified as we have seen by the action of poisons, for example, digitalis, and the products of asphyxia. It has also been seen that inhibitory (vagal) influences have a profound effect upon the transmission of impulses, (Figs. 81, 82 and 172).

The facility with which impulses are conducted is also modified in relation to the sinus rate. In a heart in which conduction is impaired, a rise of auricular rate is followed not only by an increase in the grade of partial heart-block as a direct effect of the inability of the tissues to transmit impulses so rapidly, but there is also an actual and resultant exaggeration of the hindrance.^{2 & 13} Thus an acceleration of the auricle, while conduction is normal, may be accompanied by a decrease of the *As-Vs* interval; on the other hand, and paradoxically, when the *As-Vs* interval is originally prolonged, acceleration may lead to a further widening of the interval. The disturbance may be even greater, and high grades of partial heart-block or even complete dissociation may result (Plate III, Fig. 219 and 220).

From the clinical standpoint the observations are of twofold importance. In the first instance a rise of sinus rate may unmask the lesser degrees of heart-block. In patients in whom the *a-c* interval and the *P-R* interval show little or no appreciable prolongation, the absence of reserve in the functions of the junctional tissues may be exposed by an increase in the number of impulses reaching them. Dropped beats appear in an otherwise sequential rhythm (Plate III, Fig. 220); the picture of a 2:1 ratio may be produced. Clinical examples of the effect of acceleration of the heart rate are not infrequent where, in the presence of deficient conduction, the patient is also the subject of paroxysms of tachycardia. Single dropped beats may be observed during an attack⁸ and are illustrated by Fig. 173. A higher grade of partial heart-block (a 2:1 ratio) is shown as an accompaniment of an auricular paroxysm¹² in Fig. 174 and 175.

In the second instance, a rise of sinus rate may be succeeded in experiment by prolonged periods over which there is an absence of response, so-called *standstill* of the ventricle; and if such intervals of standstill are of sufficient length, they may be accompanied by grave disturbances of the circulation.

There are several instances on record in which auricular tachycardia and a high grade of partial, or even complete, heart-block have been found in association.^{5 & 6} In these instances it may be difficult to state in what measure the degree of heart-block is the direct result of the acceleration of the auricle. It may certainly be assumed that impairment of the functions of the auriculo-ventricular tissues is present. But in such cases of complete dissociation it cannot be assumed that the tissues are incapable of transmitting impulses.

Auricular fibrillation and heart-block.

It has been said that tachycardia of auricular origin is found in conjunction with heart-block. Similarly, fibrillation of the auricle and heart-block are met with in the same subject. But as auricular fibrillation is

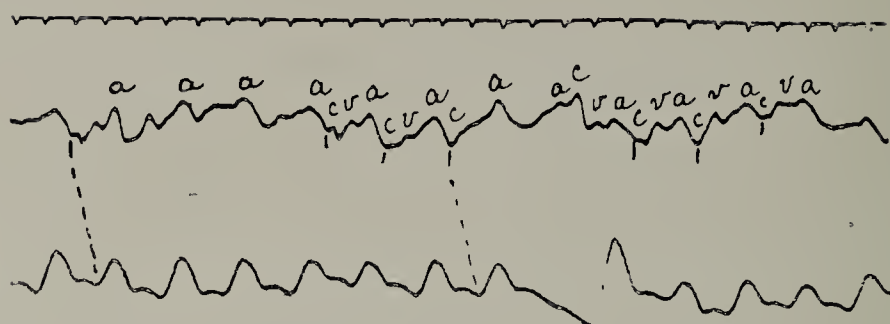


Fig. 173. (*Heart*, 1909-10, 1, 64). Polygraphic curve from a case of paroxysmal tachycardia arising in the auricle. The pulse rate is approximately 144. A single response was missed at frequent intervals. There is a shortening up of the *a-c* interval following the long pause.

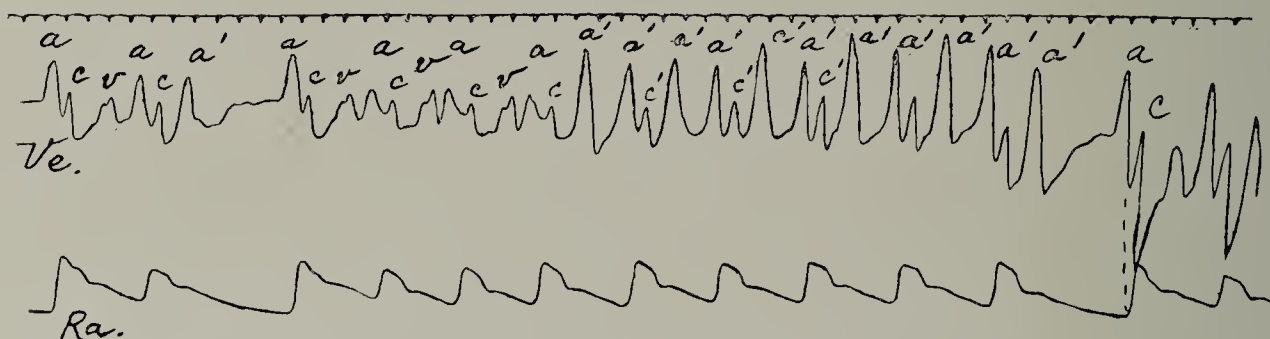


Fig. 174. A polygraphic curve from a case of paroxysmal tachycardia. The curve opens with two beats of the normal rhythm, a single premature auricular contraction (*a'*) follows, and this is succeeded by four regular cycles, each accompanied by *a*, *c* and *v* waves. From this point onwards the radial pulse shows little or no change, but the venous pulse presents signs of a tachycardia arising in the auricle, at approximately twice the rate of the normal rhythm. Each second auricular impulse is blocked. Fig. 175 is from the same case.

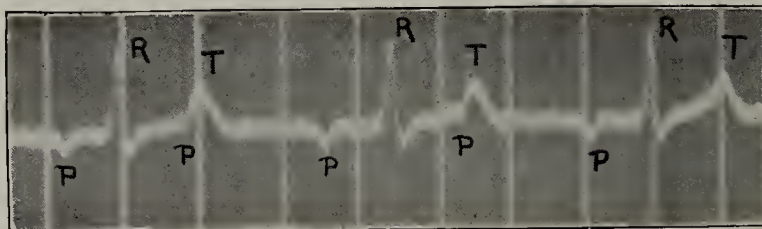


Fig. 175. (*Heart*, 1910-11, 11, 23, Fig. 12). Electrocardiogram from a case of paroxysmal tachycardia; the ventricle is responding to each second auricular impulse. The auricular complexes are inverted; alternate beats fall in presystole, and alternate beats at the commencement of *T*, notching it in the downward direction (compare Fig. 49 and 174, which are from the same case).

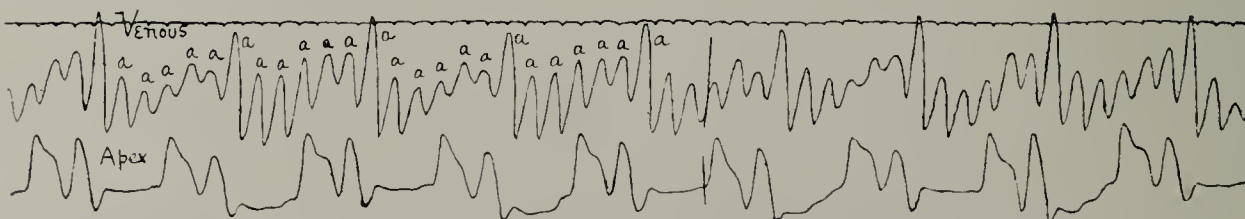


Fig. 176. ($\times \frac{5}{8}$). (Kindly lent by Dr. Mackenzie). A polygraphic curve from Hertz and Goodhardt's case.⁵ The auricle is contracting at the rate of 246 per minute. The ventricle is beating in coupled fashion (apex curve). The first beats of the couples are responses to each sixth auricular impulse. The second beat of each couple is a premature ventricular contraction. The rate of ventricular responses is 41. An example of auricular tachycardia and partial heart-block.

far more common than tachycardia, so fibrillation is a far more frequent accompaniment of heart-block than is tachycardia; consequently the clinical importance of the combination with fibrillation is correspondingly greater.

We know as an outcome of experiment that the irregular and rapid action of the ventricle, seen while the auricle is fibrillating, results from the reception of rapid and irregular impulses from the upper chamber, which is in a perpetual state of turmoil. The transmission of the haphazard impulses through the auriculo-ventricular bundle was established by Fredericq's experiment; for he found that when the bundle is broken, the auricle no longer influences the ventricular rhythm.³ The effects of injury to the junctional tissues, while the auricle is fibrillating, are precisely parallel to the effects observed when the auricle is in tachycardia. And this statement applies to instances of partial damage, just as it applies to those in which structural integrity is completely destroyed.

Now there are clinical instances, in which the auricle can be shown to be in a state of fibrillation, and yet the rate at which the ventricular beats follow each other is lower than might be anticipated. In experimental work it is the rule that when a healthy heart is under observation, the onset of auricular fibrillation is associated with a greatly accelerated ventricular rate. In inquiring into the causation of the slow or relatively slow pulse rate, a frequent phenomenon in clinical fibrillation, it becomes essential to examine all the known means of producing retardation of the ventricular rate while the auricle is in delirium. The facts are set forth under the succeeding sub-headings.

Complete dissociation and auricular fibrillation. When the auricle is fibrillating, any interference which tends to produce heart-block under ordinary circumstances, reduces the number of ventricular responses. It has been stated that complete transection of the bundle puts an end to transmission. In fibrillation it is followed by the appearance of the same slow and regular idio-ventricular rhythm, which succeeds bundle destruction when the auricle beats regularly and co-ordinately.

Several instances of the combined picture, complete dissociation and auricular fibrillation, have been encountered clinically. The first recognised example received careful study.¹¹ It occurred in a patient who exhibited Adams-Stokes' syndrome and in whom there was a syphilitic history. The action of the ventricle was in every way characteristic of that which is noted in complete dissociation of regular auricular and ventricular rhythms. The pulse was slow (Fig. 179) (usually 32 beats to the minute) and the sequence of contractions orderly. Premature ventricular contractions, interfering with the otherwise regular rhythm, were followed by pauses of the usual lengths, pauses equivalent to the intervals between rhythmic beats (Fig. 180). The fits were the result of further retardation (Fig. 203). The ventricle presented an exactly similar mechanism to that which characterises ordinary complete heart-block. Yet the same heart

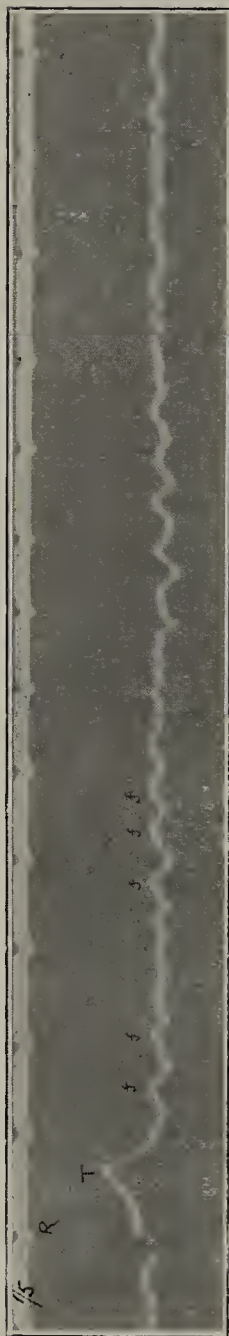


Fig. 177 ($\times \frac{1}{2}$). An electrocardiogram from a dog, in which the auricle was fibrillating and the ventricle beating very slowly and irregularly. Only one ventricular beat is shown in the figure. The remainder of the curve consists of the characteristic oscillations produced by a fibrillating auricle.

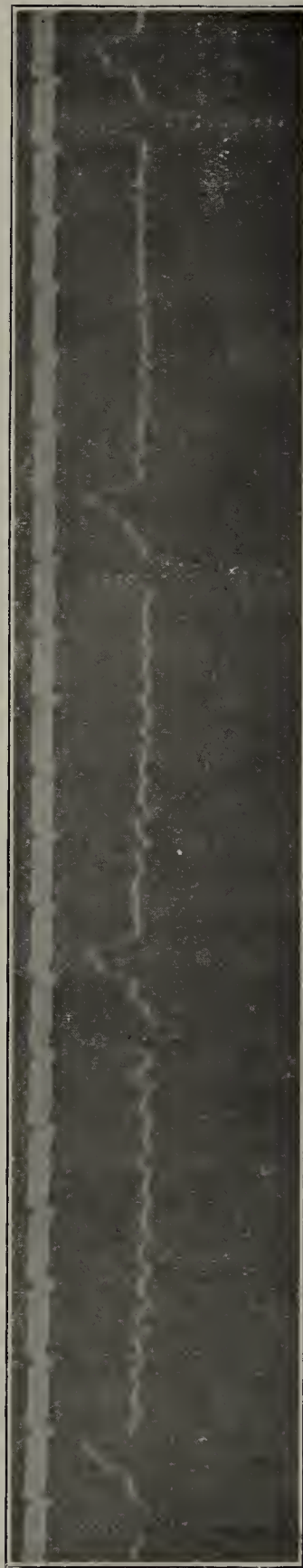


Fig. 178 ($\times \frac{1}{2}$). An electrocardiogram from a dog, in which spontaneous auricular fibrillation occurred. The ventricle beat regularly and slowly throughout. The condition may be compared with that exhibited by Fig. 179 and 182, which are from a clinical example of auricular fibrillation and complete heart-block.

manifested a striking contrast to that which displays the usual independence of auricular and ventricular rhythms. There was a total absence of all signs of auricular pulsation in the jugular curve, which was of the ventricular form (Fig. 179) and galvanometric examination established the presence of fibrillation of the auricle (Fig. 181 and 182).

Studies of the combination, complete dissociation and fibrillation, serve to emphasise the fact that fibrillation of the auricle and regular action of the ventricle do not co-exist where there is no impediment to the transmission of impulses from one chamber to the other.

Lesser grades of heart-block and fibrillation. An experimental combination of fibrillation and partial heart-block shows phenomena which are parallel to those observed when auricular tachycardia and heart-block are present together. The rate of the ventricle, as compared to its rate in uncomplicated auricular fibrillation, is slow. And if partial heart-block is already present and the ventricular rate is already retarded, a further reduction may occur when fibrillation of the auricle is induced. (Plate III, Fig. 221). The important fact from the clinical standpoint is the reduction of the expected rate, with maintenance of irregularity, when defects of conduction are associated with inco-ordinate action of the auricle. The writer has drawn attention to clinical cases reported by Mackenzie¹⁶ (under the title "Nodal Bradycardia") and to another published case,⁴ in all of which such a combination was almost certainly present. An example, taken from the series originally reported by Mackenzie, has been examined electrocardiographically, and the presence of fibrillation established beyond question (Fig. 183). The junctional tissues were known from previous observations to be the seat of a defect; the patient had exhibited a prolonged *a-c* interval for years. The case forms a good parallel with the experimental observations.

The effect of vagal stimulation upon auricular fibrillation. Stimulation of the vagus, while the experimental auricle is fibrillating has one of two effects.⁷ & ¹⁸ If the fibrillation is of short standing, it may suppress it (Fig. 186). If it is of longer duration, it usually fails to do so. The inhibitory impulses are accompanied by a certain degree of alteration in the form of the auricular mechanism itself, the movements in its walls appear to be more finely subdivided, suggesting the establishment of lines of block in the musculature. But the most conspicuous effect is a marked retardation of the ventricular rate (Fig. 184 and 185). Perfect clinical parallels of this retardation have recently been published by Wennekebach.²⁰ He describes two instances of clinical auricular fibrillation in which pressure upon the vagus in the neck caused marked slowing of the ventricle. A similar observation recently made by the writer is shown in Fig. 187. The manner in which the reduction of rate is brought about is clearly indicated where, after it has existed for a short while, the inhibitory influences abolish the fibrillation itself, for with the return of the co-ordinate auricular beats, a

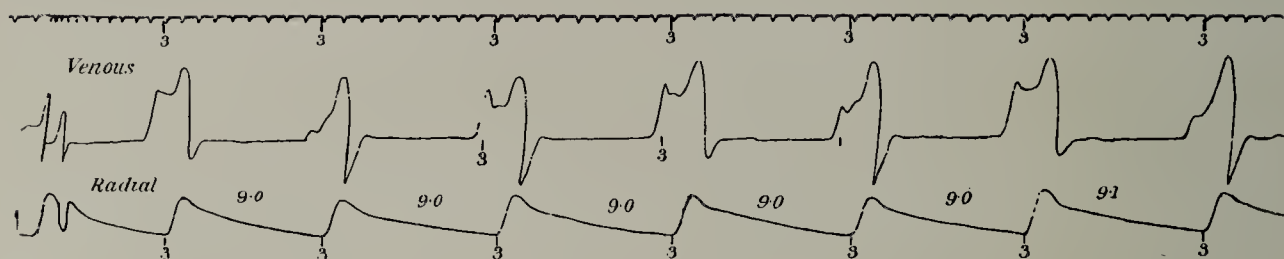


Fig. 179 ($\times \frac{5}{7.5}$). (*Quart. Journ. Med.*, 1909-10, III, 273, *Fig. 1*). Polygraphic curve from a case of complete heart-block and auricular fibrillation. (Fig. 180-182 are from the same case). The venous pulse is of the ventricular form; the radial pulse is slow and regular.

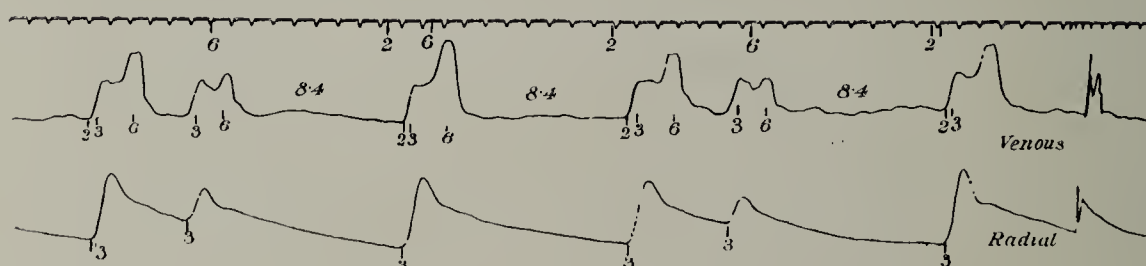


Fig. 180 ($\times \frac{4}{7}$). (*Quart. Journ. Med.*, 1909-10, III, 273, *Fig. 2*). Polygraphic curve from the same case as Fig. 179. Premature beats arising in the ventricle are followed by pauses equal in length to the pauses following rhythmic beats. This observation constituted one of the evidences of the presence of complete heart-block in the patient described. Fig. 188 is from the same case and shows the same mechanism.

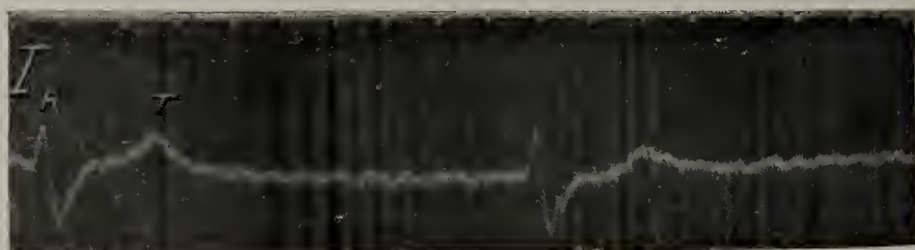


Fig. 181 ($\times \frac{1}{2}$) (*Heart*, 1909-10, I, 306, *Fig. 18*). Electrocardiogram from a case of complete heart-block and auricular fibrillation.

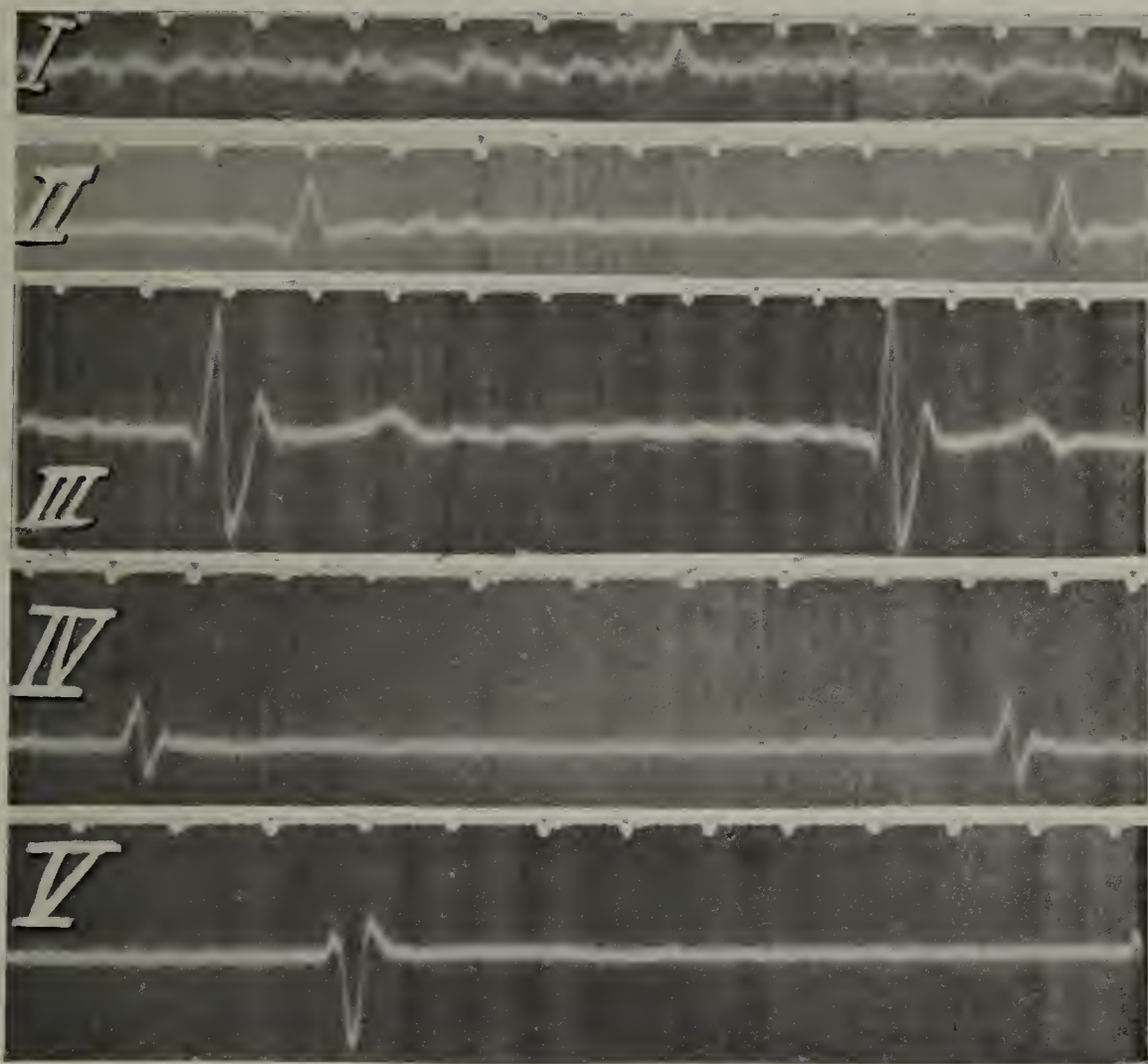


Fig. 182 ($\times \frac{2}{3}$) (*Quart. Journ. Med.*, 1909-10, III, 273, *Fig. 7*). Electrocardiographic curves from the same case; taken with special chest leads. *I*. Arm electrode at the inner end of the third right interspace; leg electrode at the inner end of the fourth right interspace. *II*. Electrodes at the gladiomanubrial synchondrosis and at inner end of the fourth right interspace, respectively. *III*. Electrodes at the gladiomanubrial synchondrosis and at the apex, respectively. *IV*. Electrodes at the outer end of the third left interspace and at the fifth left interspace in the anterior axillary line, respectively. *V*. Electrodes at the apex beat and on the abdomen below and internal to the apex, respectively.

Showing that the oscillations arise in the vicinity of the auricle and that they are independent of contraction of the ventricle.

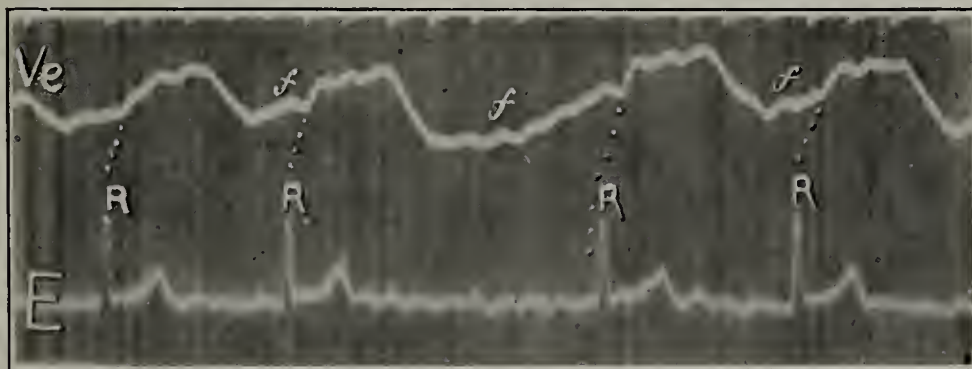


Fig. 183 ($\times \frac{2}{3}$). or (*Heart*, 1909-10 I, 306, *Fig. 19*). Electrocadiogram and venous curve from a case of auricular fibrillation, in which there was a partial obstruction to the passage of fibrillation impulses from auricle to ventricle. The venous pulse is of the ventricular form. From a case described by Mackenzie under the title "Nodal Bradycardia." Fig. 152 A is from the same case.

greater or lesser degree of heart-block is observed, (Fig. 186). The vagus as we have previously noticed may be responsible for heart-block, and there can be little question that its excitation reduces the number of responses of a ventricle to a fibrillating auricle, by establishing a hindrance to the transmission of impulses.

The clinical importance of this phenomenon, and of those which are spoken of in preceding paragraphs, lies in their relation to the rule that reduction of ventricular rate, when the auricle is in delirium, is the invariable outcome of a conduction defect, a failure of transmission of the irregular fibrillation impulses

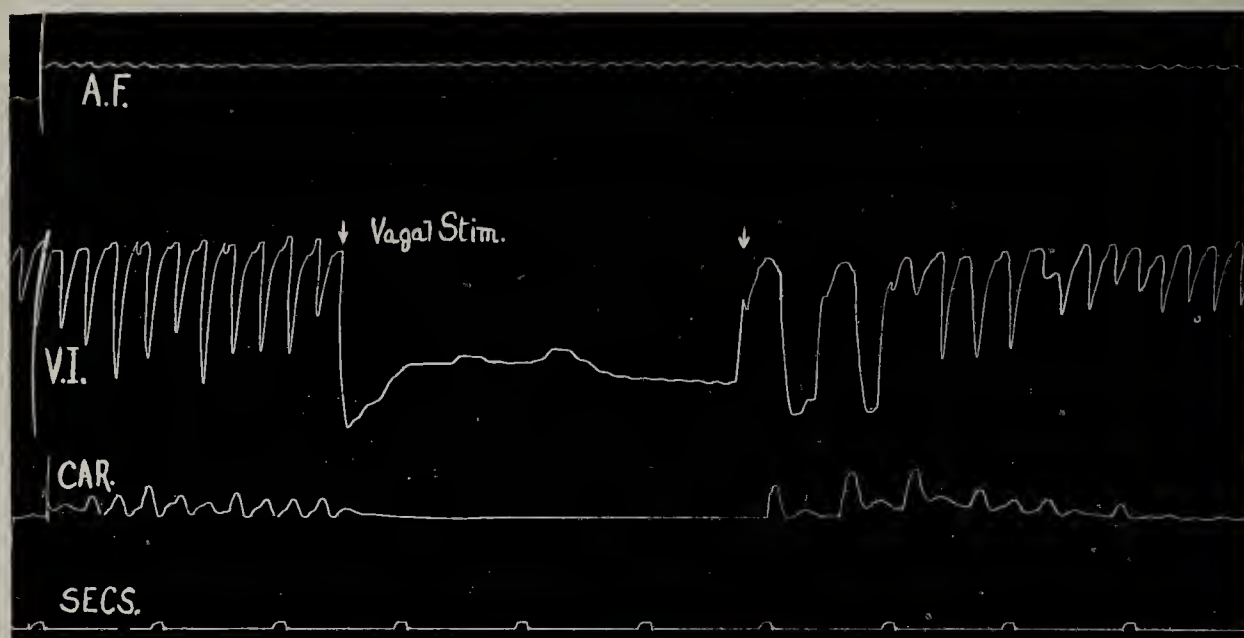


Fig. 184. ($\times \frac{1}{2}$). Myocardiographic curves (from auricle and ventricle) and Hürthle carotid pressure curve from a dog. *A.F.* the auricular curve, while the auricle is fibrillating. *V.I.* The ventricular curve, showing the irregularity of the response to the fibrillating auricle, the stoppage of the ventricle produced by vagal stimulation and subsequent recovery.

The action of digitalis in auricular fibrillation. Before finally adopting the view that slow pulse rates are found to consort with auricular fibrillation only when there is a hindrance to the passage of impulses, the question of digitalis slowing remains for consideration. We have already strong presumptive evidence for the view. The conclusion that digitalis acts in a similar manner rests upon our knowledge of the action of this drug. We are aware that digitalis has a powerful effect upon the vagus, that vagal stimulation inhibits impulse conduction, and that digitalis produces heart-block experimentally. We have also seen in an earlier chapter that its administration may be followed by the appearance of a higher grade of partial heart-block in clinical cases. It is upon the heart which manifests auricular fibrillation that digitalis exerts one of its characteristic and valued actions, namely, slowing of the ventricle.¹⁵ It is in these cases that the pace of the ventricle is often wonderfully regulated by the dosage employed. We

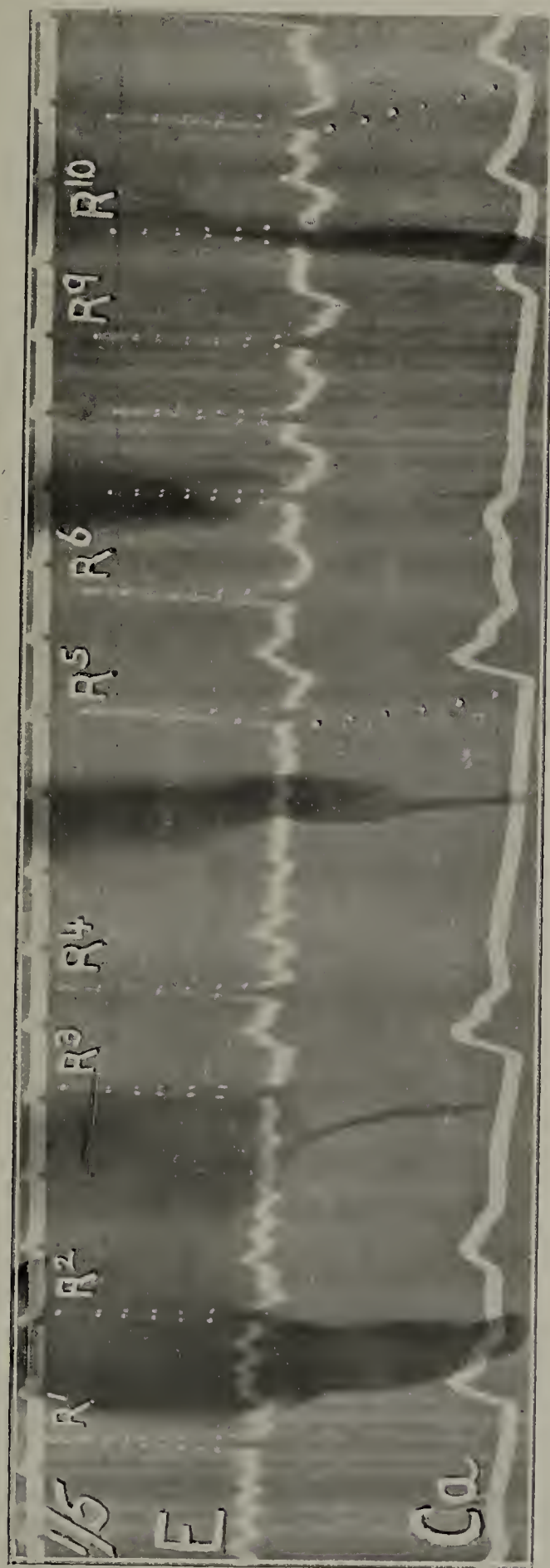


Fig. 185. (*Heart*, 1909-10, 1, 306, *Fig.* 17). Electrocardiogram and Hürthle carotid pressure curves from a dog. Over the early cycles of the figure, the heart is escaping from vagal stimulation. With the quickening of the ventricle the fibrillation oscillations are masked. Note the discrepancy between the heights of the peaks *R* and the upstrokes of the carotid curve.

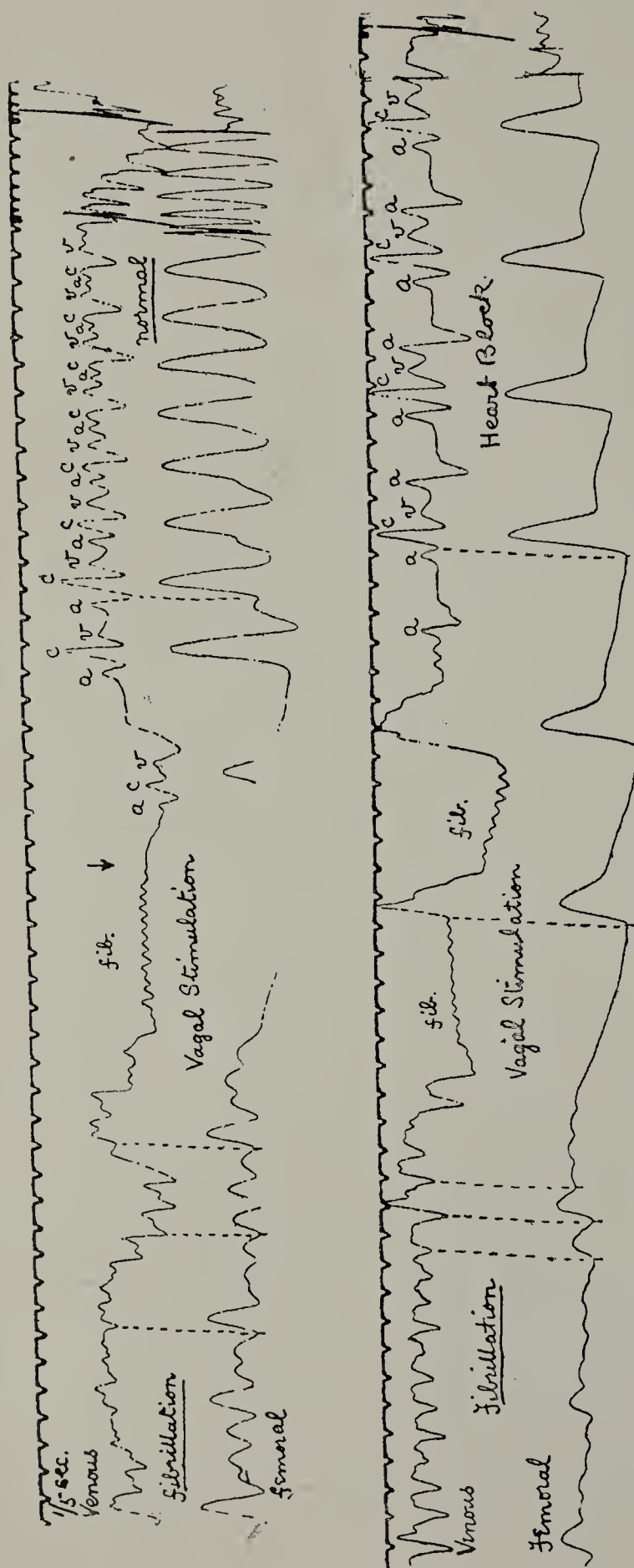


Fig. 186. (*Heart*, 1909-10, I, 344). Two polygraphic curves from a dog. Chest wall intact. Showing venous and femoral curves while the auricle is fibrillating, and the effect of vagal stimulation. At the commencement of each curve the arterial tracing is completely irregular and the venous pulse is ventricular in form. Vagal stimulation slows the ventricle and unmasks small venous oscillations, the result of the auricular fibrillation. The inhibition breaks down the fibrillation at a latter point, and with the return of the co-ordinate contractions of the auricle, heart-block is observed. The heart-block in the first figure is seen as a prolongation of the *a-c* interval, in the second figure as a 2:1 ratio. The presence of heart-block at this stage is used as evidence that the slowing during the fibrillation stage is also the result of an impediment to the passage of auricular impulses.



Fig. 187. A radial pulse curve from a clinical case of auricular fibrillation, showing the effect of pressure upon the right carotid sheath and vagus nerve. The pressure was applied and released at the points marked by arrows. The pulse, which had previously shown no pause greater than 1.2 sec., exhibits a pause of 3.4 sec., which is followed by slow ventricular action and a gradual return to the original rate. The observation was repeatedly made upon the same patient.

can arrive at no other conclusion but that its influence is exerted in the manner suggested.⁹ And the conclusion is maintained by a consideration of the type of patient in which the reaction is most marked. It is, *par excellence*, in instances of heart disease which may be attributed to a past infection (rheumatic or choreic), that the dropped beats and 2:1 ratios follow the employment of the drug; it is in the same type of heart that digitalis slows the ventricle when fibrillation is present.

But, given in clinical cases, digitalis fails to produce heart-block in the normal subject. The reaction, as has been shown,¹⁵ occurs to perfection where a pre-existing defect of conduction can be demonstrated. Again, in patients who are the subjects of fibrillation, digitalis may fail to slow the ventricle, and probably for the reason that the functions of the junctional tissues are primarily intact. In most instances in which careful observations have been undertaken before and after the onset of fibrillation,* it has been found that, according to the presence or absence of demonstrable transmission defects during the period of sequential contractions, the ventricle reacts or fails to react by slowing, during the stage of fibrillation. The proposition therefore takes the final form that digitalis slowing in auricular fibrillation is due to the effect of the drug in increasing a pre-existing defect in the transmission of impulses from auricle to ventricle.^{10 & 11} Whether it acts directly upon the junctional tissues, indirectly through the vagus, or partly in one manner and partly in the other, is not fully decided. Recent observations by Mackenzie,¹⁷ upon the influence of atropine upon the retardation, seem to show that the latter is produced at least in part, in some cases wholly, through the vagus.

We have spoken throughout of digitalis, but precisely similar statements apply to the closely allied remedies, strophanthus and squills.¹⁹

The degree of slowing produced by one or other of these drugs may be remarkable, and several instances have been observed in which the ventricular rate has been reduced to an extent compatible with complete dissociation; it may become so slow that its rhythm is absolutely regular.¹⁶ These effects may be compared with the observation that where the auricle is contracting co-ordinately, the administration of drugs of the digitalis group can lead to the establishment of complete auriculo-ventricular heart-block.

The actions of digitalis upon the human heart, when the auricular contractions are co-ordinate and when the auricle is fibrillating, are compared in the accompanying table with the several grades of spontaneous heart-block observed under similar circumstances.

* Such cases are few in number.

A-V RHYTHM.		AURICULAR FIBRILLATION.	
<i>Heart-block.</i>	<i>Digitalis, H. B.</i>	<i>Heart-block</i>	<i>Digitalis H. B.</i>
Lengthened <i>As-Vs</i> interval.	Lengthened <i>As-Vs</i> interval.	Spontaneous slowing of irregular tachycardias. ¹⁵	Ordinary digitalis slowing.
2:1, 3:1, etc. Heart-block.	2:1, 3:1 Heart-block. ¹⁵	Spontaneous slowing of marked grade, ¹⁶ and cases with onset of slow and irregular rhythm. ⁴	Marked slowing on digitalis. ¹⁵
Complete Heart-block.	Complete Heart-block.	Auricular fibrillation and complete heart-block, (Fig. 179).	Digitalis slowing with the production of a regular pulse of 30-40 per minute. ¹⁶

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CHAPTER XX.

AURICULAR FIBRILLATION AND HETEROGENETIC BEATS OF VENTRICULAR ORIGIN. SO-CALLED "BIGEMINY."

Auricular fibrillation and heterogenetic beats of ventricular origin. The coupled beats of digitalis.

It is well known that if one auricle is thrown into a state of fibrillation experimentally, the other follows suit. The same phenomenon is observed in fibrillation of the ventricle. A limited portion of the intact auricular or ventricular tissue cannot be maintained in fibrillation while the remainder beats co-ordinately; the condition spreads and involves either the whole auricle or the whole ventricle. Yet it is recognised that fibrillation is never transmitted as such from auricle to ventricle or from ventricle to auricle. When the auricle is fibrillating, the ventricle responds to the impulses received from it in an irregular but nevertheless co-ordinate manner. Often, when the ventricle fibrillates, the auricle likewise beats arrhythmically, but with co-ordination of the separate contractions. These phenomena suggest that fibrillation can spread from one area to an adjacent area only when the bridge of tissue which effects the union between them is so constituted and arranged that it may participate in the process. The auriculo-ventricular bundle, which transmits the impulses from auricle to ventricle while the former is in delirium,¹ is sharply defined on all sides. Continuity of the musculature of the two chambers is established at the terminations of the junctional tissues above and below, and at these points alone. The union is by a narrow band. Consequently, while the auricle fibrillates, the bundle transmits only certain of those impulses which are showered promiscuously upon the small area abutting upon the upper termination (the auriculo-ventricular node). Contraction waves flowing in several directions towards the narrowing channel, which forms the impulse-inlet to the ventricle, are eventually transmitted in a single direction (along the usual path), and confusion of impulse transmission in the bundle itself is avoided. From the turmoil in the auricle a rapid and haphazard succession of waves escape, and, escaping, are confined to a single course, the boundaries of which are parallel. Thus two facts are explained: first, the absolute irregularity of the ventricular responses to a fibrillating auricle, and second, the inability of the fibrillation to transmit itself from upper to lower chamber or *vice versa*.

But regarded in this way, and assuming the function of the junctional tissues to be perfect, it will be obvious that when auricular fibrillation is present it is almost if not quite inconceivable that the ventricle should beat

regularly in response to impulses generated in a chamber in which the inco-ordination is of a high grade.* Nevertheless, in the condition to be considered, it does not infrequently happen that certain of the pauses preceding pulse or heart beats are of constant length.

A state of accurate coupling of ventricular beats may occur, and is a frequent, though not exclusive, result of the administration of drugs of the digitalis series. This coupling is illustrated by the accompanying figures (Plate IV and V, Fig. 224 and 225). It is of a curious nature, for while the distance from large to small beat is constant, that from small to large beat shows the same want of regulation as do the intervals between adjacent beats of the uncoupled rhythm. If the auricle is known to be fibrillating, the accurate coupling under consideration evidently requires further elucidation. The ventricular electric complex of the normal beat is characterised in its opening phases by a quick movement in the upward direction. The direction and shape of this peak (*R*) are indications of the supraventricular origin of the ventricular contractions. If in a curve composed in the main of individual ventricular complexes of the normal type we meet with other complexes of a divergent type, we may suspect that the last have arisen in a different manner. Now the electro-cardiographic curves which are obtained from patients with complete irregularity of the heart exhibit individual complexes which are for the most part normal in general outline. That is to say, the electric complexes establish the supraventricular derivation of the contractions represented. It also happens that other types may be mixed with them.^{2, 3 & 5} These divergent types are preceded by shortened pauses, and are always absolutely or relatively abortive in their efforts to raise the arterial pressure (Plate IV, Fig. 222). The divergent type of beat is usually scattered at irregular intervals along the curves, but when more frequent it may occur after each beat of normal type, and at such times may produce the picture of accurate coupling previously mentioned.

The divergent types of beat are due to heterogenetic impulses arising in the ventricle itself, and are of precisely the same nature as are the premature contractions, which, originating in the ventricle, disturb a normal and regular sequence.⁵ And this can be clearly shown in an individual patient by a comparison of the ventricular interruptions before and after he becomes affected with fibrillation of the auricle.⁶ The record of the interrupting beat is precisely the same whether it is taken before or after the onset of fibrillation. Heterogenetic beats arising in the ventricle and complicating the irregularity produced in this chamber by the haphazard response to fibrillation impulses are extremely common. They have been found in twenty cases of a total number of sixty examined.

*Were the inco-ordination less it might be possible that an irregularity would result in the ventricle, which recurred periodically; thus we might be led to anticipate, under such circumstances, an occasional or a rhythmic duplication of a particular period of irregularity. Such repetition is not encountered in the abnormal mechanism considered, and we are therefore forced to assume that the generation of impulses proceeds in a highly disorderly and changing fashion.

A polygraphic tracing including venous and radial curves is shown in Plate IV, Fig. 224. The central cycles of the figure belong to a period of bigeminy, and the earlier and later ones to the complete irregularity in its uncomplicated state. The venous pulse is of the ventricular form. Each of the larger beats in the radial curve is accompanied by two systolic waves in the jugular curve. The smaller beats of the bigeminy are represented by single waves in the jugular curve. The distinction between strong and weak beats is even clearer in the electric curve (Plate V, Fig. 225). The beat which is preceded by the longer or inconstant pause is of the usual type; the complex starts with a peak *R* directed upwards and of short duration. The beat which follows and corresponds to the smaller radial pulsation is of a divergent type. The mechanism with which we have to deal is therefore perfectly apparent. The auricle is fibrillating and each alternate beat of the bigeminy is the result of a haphazard auricular impulse. The beats of auricular origin are followed at a constant time interval by beats of intrinsic ventricular origin, for they conform to a type recognised as the outcome of premature ventricular beats interrupting an otherwise normal rhythm. Plate V, Fig. 226, another example, demonstrates a type of premature ventricular contraction which is more readily identified as emanating from the apical and left portions of the ventricle. Only a short strip of curve intervened between Fig. 226 and 227 (Plate V), yet the type of divergent beat has altered, though the spacing of the couples at $\frac{2.6}{5}$ sec., remains constant. The premature contraction in this instance (Fig. 227) approaches a type of beat, yielded experimentally in the dog by stimulation of the right ventricle at a point not far distant from the middle of the inter-ventricular furrow and on the anterior surface.

So-called 'bigeminy' of the heart.

Wenckebach, ^{7 & 8} in discussing bigeminy of the heart, separated what he regarded as a special cardiac arrhythmia under the term 'true bigeminy.' He clearly recognised that the picture of paired beats arises in many instances as a result of premature contractions, each falling subsequent to a ventricular systole having its origin in an auricular impulse. He regarded such examples of paired beats as lying outside a rational definition of bigeminy, and desired to restrict the term to instances of twin beats which present certain definite characteristics. The qualities of an isolated couple of beats, which he deemed essential in order that it might be brought within the limits of the new definition, were two in number. First, that the second beat of the pair should lack a complete compensatory pause, and secondly, that the two beats should bear a constant time relationship to each other. In a later publication the definition was extended by the exclusion of the first qualifying factor. In brief, the definition was to include all instances of accurate coupling. In dealing with the subject Wenckebach distinctly implies that the term bigeminy, to be

logically employed, must be restricted to such of the twin beats as may be supposed to be constituted by individual beats of an identical nature. Such an implication involves the assumption that where beats are accurately coupled, the pairs consist of individual beats of an identical nature.

We have seen that in cases of complete irregularity of the heart in which, from time to time, phases of accurate coupling or bigeminy are prominent, the mechanism is attributable to the alternate response of the ventricle to impulses derived from auricle and ventricle respectively. The curves were taken from cases actually or recently under the influence of digitalis. The bigeminy seen in similar cases in the absence of drug administration is of an essentially similar nature. The beats preceded by the constant or shortened pauses are of a divergent or anomalous type.

To what then is the accurate coupling attributable? It has been suggested that the first beat is actually the cause of the second. This view is supported by the constancy of the pause and is further evidenced by such an instance as that shown in Fig. 226 and 227 (Plate V), where without alteration in its time relationship to the preceding beat, the divergent type of contraction arises now from one point and now from another point in the ventricle.

Amongst the examples of true bigeminy cited by Wenckebach, one of the most prominent was that of accurate coupling of contractions in complete auriculo-ventricular dissociation. An example of the condition is shown in the accompanying illustration (Plate IV, Fig. 223). The figure demonstrates:—(1) three curves, of which the upstroke of each is marked by the letter *R*; they represent ventricular beats which belong to the ventricular rhythm proper; (2) three premature beats (each consisting of two main variations *Ep* and *En*) coupled at a constant time interval to a preceding beat of the ventricular rhythm; (3) and finally, superimposed upon the whole curve, a regular succession of auricular waves, marked *P*. The length of the pause following the extra beats varies slightly, but approximately corresponds to the pause between beats of the uninterrupted ventricular rhythm (the rate of this rhythm was 32 per minute on the same day). We are in the presence of an example of idio-ventricular rhythm disturbed by a regular succession of abnormal ventricular contractions, all of a similar nature. It is believed that the beats of the ventricular rhythm proper have their origin at the point from which the ventricular wave of the normal heart-beat starts. The interrupting beats are premature contractions arising in the ventricle, and they are of a variety such as can be shown, experimentally, to spring from an area of ventricular musculature lying to the left and in the neighbourhood of the heart's apex. The ventricular arrhythmia, as a whole, is therefore recognised as resulting from the alternate appearance of beats arising from separate foci in the ventricle.

But similar 'bigeminies' are occasionally found in which the first and second beat of each couple yield identical galvanometric curves; for example that depicted in Fig. 188. The rhythm dominating the ventricle

in this patient was of the idio-ventricular type; its rate was approximately 32 per minute. It is interrupted by premature beats (the second of each couple) which arise at the same focus. It is an example of an irregularity

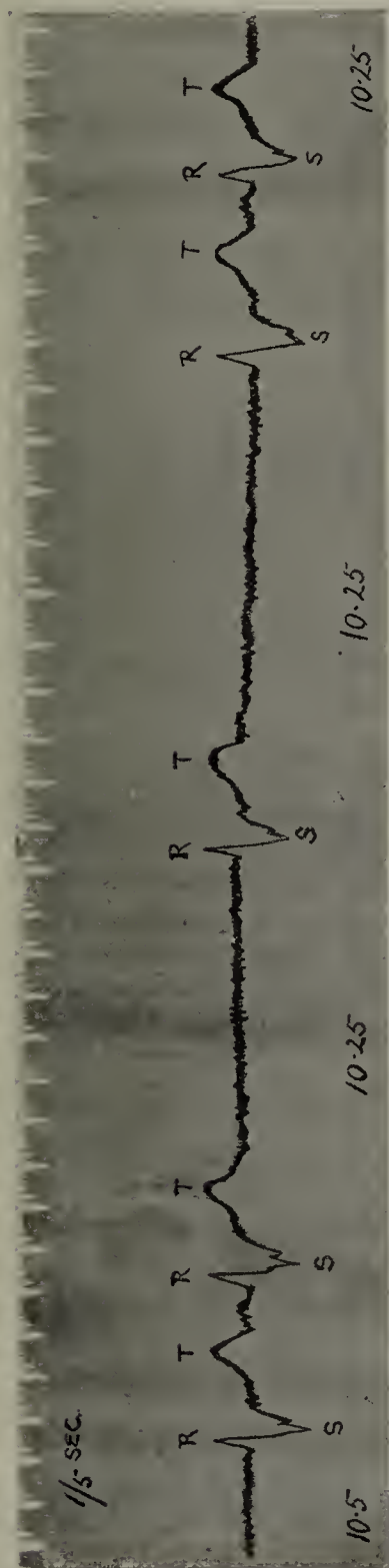


Fig. 188 ($\times \frac{6}{5}$) (*Quart. Journ. Med.*, 1909-10, III, 269, *Fig. 2*, and *Lewis and Mack, ibid.*, 273, *Fig. 6*). Electrocardiogram from a case of complete heart-block and auricular fibrillation. Showing coupled beats, in which the first and second beats of the couple arise at a single point, (known from the similar outline of the complexes); a rare occurrence. The pause following the second beat of a couple is equivalent to that which succeeds a rhythmic beat; the usual phenomenon in complete dissociation.

similar to that seen in Plate IV, Fig. 223; but in the second instance both the first and second beats of the couple arise from the point at which the idio-ventricular starts. The fundamental ventricular rhythm may therefore be regarded as the same in each case, and the difference between them (Plate IV, Fig. 223 and Fig. 188), as evidenced by the galvanometric curves, lies in the region from which the interrupting beats arise.

If the term 'true bigeminy' is taken to imply that the first and second beats of a couple are of an identical nature, the first example (Plate IV, Fig. 223) obviously lies outside the definition. In regard to the second example, it may certainly be argued that the impulses of the couple arise at the same focus, and that the contraction wave travels through the ventricular musculature in the same direction in each individual beat of the pair. But that the two beats are in every respect of an identical nature remains to be proved. The fact that the one is invariably preceded by a longer and the other by a shorter pause is in itself sufficient to demonstrate that in their production there is an essential difference. The variation in the length of preceding pause is indeed a fundamental distinction. In both the examples figured we are dealing with a single phenomenon, the interruption of a slow (ventricular) homogenetic rhythm by heterogenetic beats. The sole distinction lies in the fact that in the first instance the

heterogenetic systole happens to arise at a point some distance from the ventricular pace-maker, while in the second instance it arises in its vicinity.

When beats are linked together at accurate intervals for a considerable time, it is probable, as Wenckebach suggests, that the second beat of the pair is the direct result of the first. It might be convenient to confine the term bigeminy to arrhythmias in which this phenomenon is supposed to exist. But here also a difficulty arises. Irregularities in which beats are coupled over long stretches of curve, but in which the coupling is not quite accurate, are well known. There is every possibility that in these instances also, the second beat of a couple is the offspring of the first, and there appears to be no sufficient reason for a sharp distinction between the mechanism of production in one case and the other, when it is regarded from this point of view.

It consequently seems advisable that the term bigeminy, employed in any sense other than that of the coupling of beats, should be allowed to lapse ; at all events, until the time when we are in fuller possession of the facts in regard to the irregularities which we have been considering.

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CHAPTER XXI.

THE VAGUS.

The vagus nerve has been the subject of numerous physiological researches^{1 & 11} since the discovery of its inhibitory functions by the brothers Weber. Gaskell, (and later Engelmann) utilised it in his endeavour to demonstrate the isolation of certain cardiac functions. We are more especially concerned with those reactions of the heart to vagal influences which are encountered in clinical work, and with the known effects of the vagus upon the heart which have a direct and practical bearing.

Respiratory arrhythmias.

Respiratory arrhythmia in experiment. In the dog and cat, placed under experimental conditions, there is, as is well known, a definite alteration in the lengths of the diastolic pauses of the heart with the several phases of respiration. The relationship is such that with natural breathing the pauses increase in length in expiration and decrease with inspiration. The longest beats occur when intrathoracic pressure is highest, the shortest beats when intrathoracic pressure is lowest, Fig. 189. Section of the vagi, or saturation with atropine, abolishes the respiratory variation entirely.

The degree of respiratory arrhythmia accompanying natural breathing varies greatly from one animal species to another. It is very conspicuous in the dog and occurs even during sleep, when respiration is slow and shallow (Fig. 190).

Respiratory arrhythmia in the healthy human subject. In the adult man, respiratory variations of pulse rate are either little in evidence or entirely absent while the breathing is natural. But in children their presence is rather the rule than the exception, and they constitute the chief source of irregular heart action met with in very young subjects.

At or about the epoch when the pulse diminishes in rate and the heart assumes the rhythm which it will maintain during adult life, it may be well

marked, and occurring at or about the time of puberty, has been termed "Youthful irregularity" by Mackenzie,¹⁰ (Fig. 192).

A periodic arrhythmia associated with forced breathing is universal (Fig. 191) and the type of irregularity and its relationships to respiration are the same as those already mentioned as happening in the experimental animal.

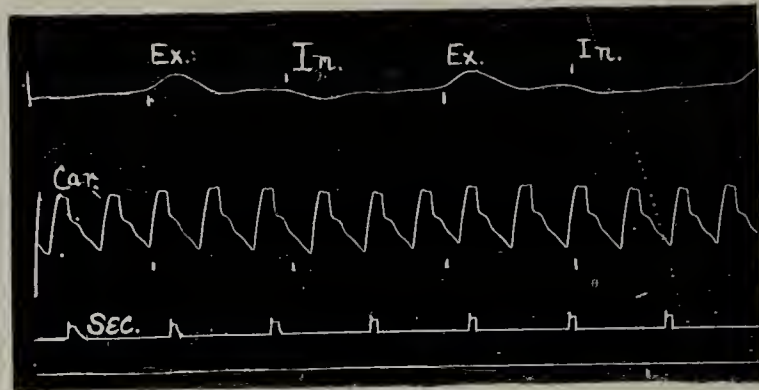


Fig. 189 ($\times \frac{2}{3}$). Simultaneous intratracheal pressure and Hürthle carotid pressure curve from a cat, breathing naturally. With each normal expiration the intratracheal pressure rises, with each normal inspiration it falls. Raised intrathoracic pressure is accompanied by a slowing, and lowered pressure by a quickening of the heart beats. Time in seconds.

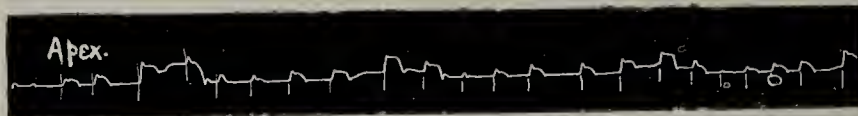


Fig. 190 ($\times \frac{2}{3}$). An apex tracing from a terrier, taken during sleep and while the breathing was slow and shallow. The periodic waxing and waning of ventricular rate is well marked.



Fig. 191 ($\times \frac{2}{3}$). (*Journ. of Physiol.*, 1908, xxxvii, 252). Polygraphic curve from a young adult. Simultaneous pulse and respiratory curves. There is little or no alteration of mean blood-pressure, but a conspicuous variation in the length of the pulse intervals. Lowered intrathoracic pressure (the summit of inspiration) is associated with the shortest, and raised pressure with the longest beats.

The almost constant relationship of these arrhythmias to the separate acts of respiration stamps them as vagal in origin; a conclusion which is borne out by their disappearance with increased pulse rate.

Respiratory arrhythmia, associated with pathological processes. While it is difficult, if not impossible to draw a hard and fast line between the physiological and pathological phenomena, in dealing with respiratory changes



Fig. 192 (Mackenzie, "The Study of the Pulse." Fig. 166). Simultaneous tracings from the chest wall and radial artery. The irregularities in the pulse correspond to the movements of respiration. When the breathing is suspended the irregularity persists for one period. An example of "Youthful irregularity."

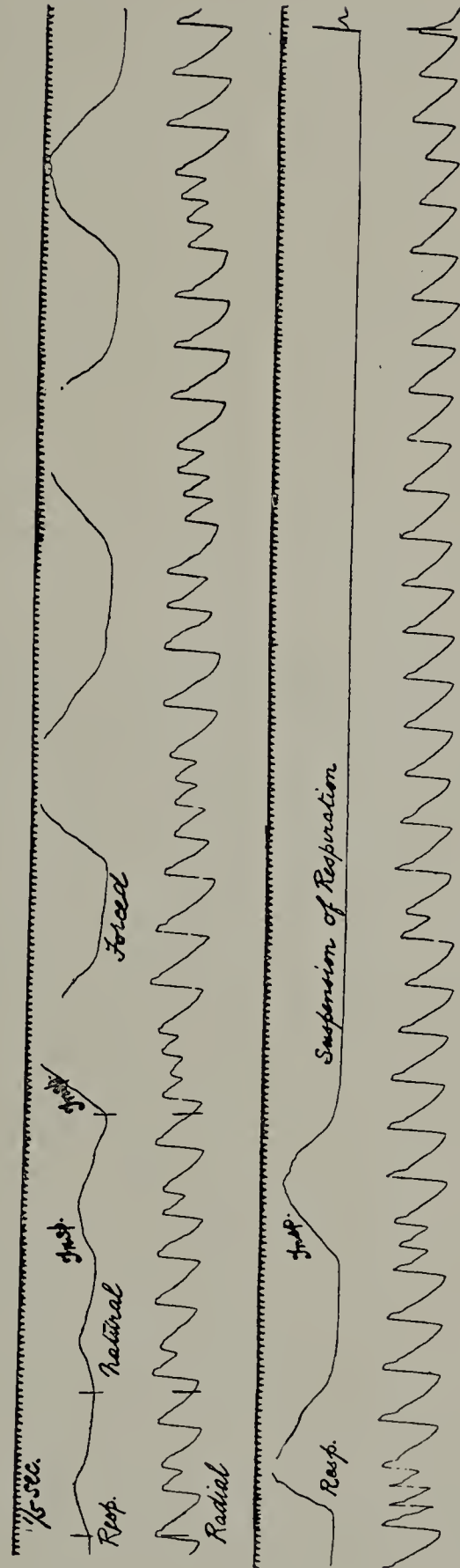


Fig. 193 *a* and *b* ($\times 1\frac{1}{2}$) (Heart, 1909-10, I, 300). Respiratory arrhythmias observed in a case of angina pectoris. The first curve shows the marked quickening with natural and forced inspiration. The second curve demonstrates the almost complete dependency of the irregularity upon breathing. The pulse becomes regular when respiration is suspended. A single irregularity is noted during the period of apnoea. The whole heart was involved in the irregularity (cp. Fig. 194).

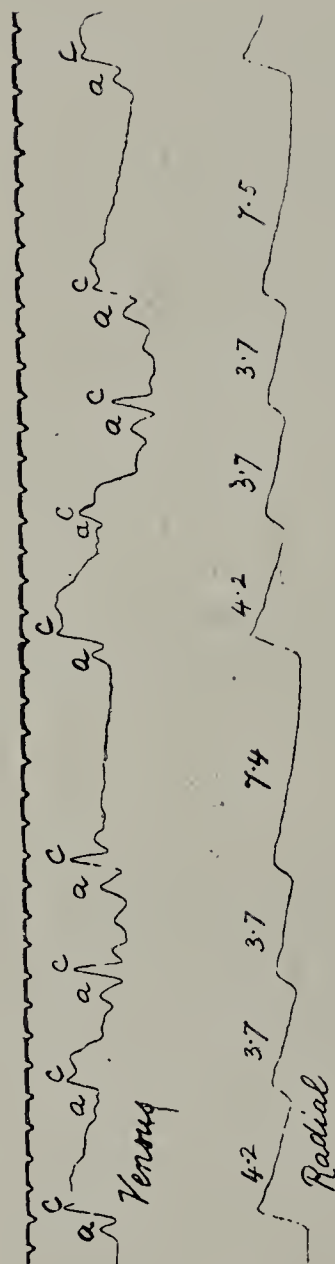


Fig. 194 (*Heart*, 1909-10, I, 303). Polygraphic curve showing long and periodic pulse pauses. The whole heart is involved in the irregularity, which was dependent upon respiration. From the same case as Fig. 193.

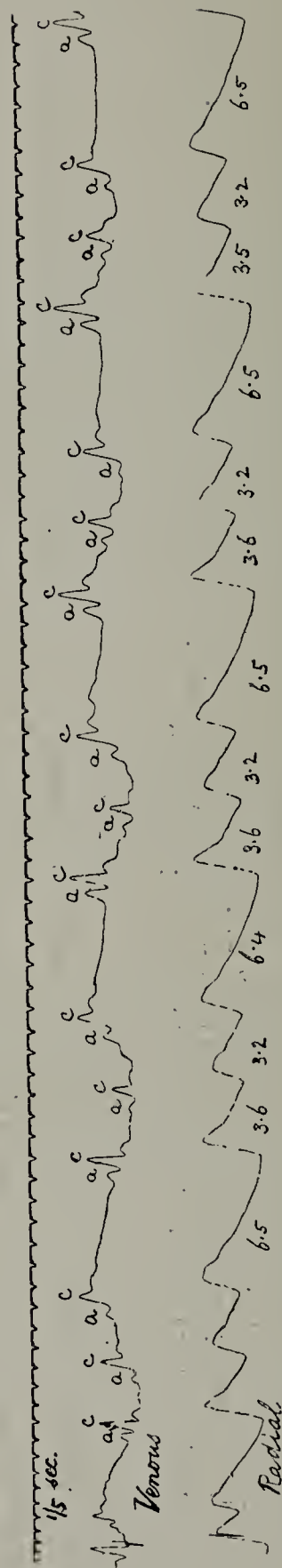


Fig. 195 ($\times \frac{7}{5}$) (*Heart*, 1909-10, I, 301). A polygraphic curve showing a similar irregularity. Respiration is shown in the venous curve. The long pauses occur at the height of expirations. From the same case as Fig. 194.

of pulse rate, it may be said that the variations are peculiarly prominent in certain pathological states.

It is known that marked changes of pulse rate, as accompaniments of *natural* breathing, are seen during convalescence from acute illnesses. They are also met with in an exaggerated form in other and less readily defined conditions. Patients may be found in whom, with each act of normal expiration, the ventricular rate diminishes to one half, to increase once more with the succeeding inspiration.¹⁸

These arrhythmias are often so pronounced in degree that they cannot escape attention. It is a rule that they are accompanied by a general reduction of pulse rate; the prominence of the arrhythmia is clearly related to the mean vagal tone exerted at the time when the observations are made (Fig. 193-195).

Irregularities produced by vagal stimulation in animals.

When the vagus is stimulated experimentally, the resulting change in the mechanism of the heart beat is very variable. We may conveniently

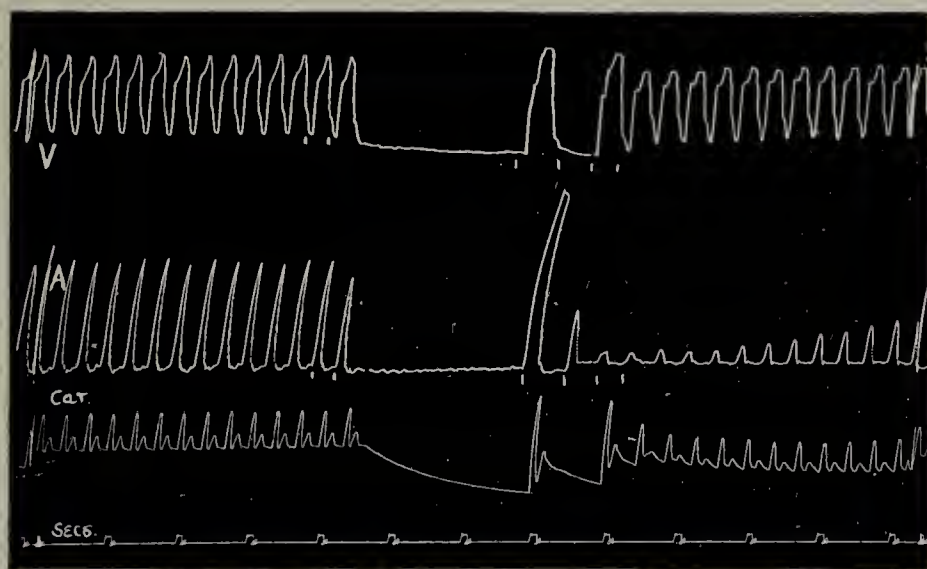


Fig. 196 ($\times \frac{1}{2}$). Myocardiograms from the auricle (A) and ventricle (V) and Hürthle carotid pressure curve (C), showing the effect of vagal stimulation. There is a sudden standstill of the whole heart. The auricle escapes first and is responded to by the ventricle after an increased auriculo-ventricular systolic interval. The next auricular contraction meets with no response. The rate gradually quickens as the heart escapes.

group the more important effects under four headings, and the separate phenomena may be compared to the clinical findings, which are described subsequently.

(i). *Slowing.* When the excitation of the inhibitory nerve is weak in its intensity, the usual effect is a retardation of the rate of the whole heart. The transition from faster to slower and from slower to faster rhythm is gradual. There is but rarely a sudden step from one rate to the other,

but the change takes place during the course of a few heart beats, and this applies more especially to the offset of the slowing. A clinical parallel is found in certain instances of phasic variation of pulse rate.

(ii). *Standstill of the heart.* When the excitation is stronger it may lead to standstill of the whole heart. By the term standstill we mean a *sudden* increase of the diastolic pause, and the production of a quiescent interval, the length of which is out of all proportion to the duration of the immediately preceding pauses, (Fig. 196). Recovery from the standstill is usually rapid and may be accompanied by the phenomena described in the ensuing paragraphs.

(iii). *Hindrance to the passage of auricular impulses to the ventricle.* The relationship of the nerve to the conveyance of impulses across the auriculo-ventricular junction has been considered in previous chapters, (cp. Fig. 81, 82 and 172).

(iv). *Escape of the ventricle.* In the majority of instances of experimental vagal stimulation, where standstill is induced, a period of slow and irregular action of the heart follows upon the longest pause. Myocardiograms taken from the auricle and ventricle, or electrocardiographic curves taken from the intact animal, demonstrate that the irregularity and the many variations met with in it are due to the interference of several factors. At the end of the standstill the auricle may escape first and one or more of its systoles may be recorded before the ventricle contracts. On the other hand, the ventricle may respond, but the intersystolic intervals may be prolonged and variable. Not infrequently the ventricle elaborates impulses of its own, and this is more especially the case when it receives no impulses from the auricle for a prolonged interval. It is obvious that auricle and ventricle may escape together and the first beat of the returning rhythm may be constituted by a simultaneous contraction of auricle and ventricle.

We may now proceed to examine the clinical facts which can be brought into line with these experimental findings.

Examples of vagal phenomena as they occur clinically.

Phasic irregularity independent of respiration. As an accompaniment of respiration in the adult subject, periodic increase and decrease of pulse rate is readily recognised as the outcome of co-existent decrease or increase of vagal tone. But it happens that irregularities of a clearly parallel character occur apart from breathing. In taking a continuous curve from such a subject it may be found that from time to time a conspicuous phase of pulse slowing occurs (cp. Fig. 26 and 198). During the course of a few heart beats the diastolic pauses increase very greatly in duration, and succeeding the largest intervals is a return, spread over a few beats, to the normal rate.^{8 & 22}

It is a type of irregularity especially associated with poisoning by members of the digitalis group, drugs which are known to exert a powerful influence upon the vagus.²⁰ Where a predisposition exists, it has been induced by pressure upon the vagus in the neck.^{8 & 22} In some patients it is peculiarly prominent when the pulse slows after its rate has been accelerated by exercise.²⁰ It may be accompanied by other symptoms which will be noted subsequently.

Compression of the vagus in the human subject. It has been shown that inhibition of the human heart in healthy subjects may be brought about by pressing deeply into the tissues of the neck, (Czermak,⁴ Waller).²¹ The procedure meets with success more frequently upon the right side than upon the left. Firm pressure is exerted with the fingers or thumb directly over the carotid in the middle or lower third of the neck, and the artery may or may not be obliterated before the slowing of the heart is observed. Vagal compression is not without risk. Thanhoffer¹⁹ states that he took tracings from students who pressed upon their own vagi. Bilateral compression was utilised in the experiments. He observed standstill of the heart in one instance for a period exceeding a minute, and unconsciousness supervened. In this particular experiment serious after effects were noticed. Quincke¹⁵ undertook a large series of observations and obtained a positive result in 47 out of 80 individuals (14 positive effects were observed in 20 healthy students). Quincke illustrates his first article by several radial curves in which marked ventricular slowing or standstill is apparent.

Compression of the vagus was attempted by Rihl¹⁶ in a case of partial heart-block and, an increased grade of block resulting, a marked slowing of the ventricle and syncope was the outcome. In auricular fibrillation it has been shown to produce ventricular slowing.

Vagal excitation has also been adopted as a means of checking paroxysms of tachycardia,³ and met with conspicuous success in Bensen's case; but as a general rule it is of no avail.

Instances of sudden death following operations upon the neck in which the vagus has been included in a ligature are known to surgery and to experiment, and apparent examples of both have come within the personal experience of the writer.

Standstill of the heart as a clinical phenomenon. Some years ago, a case was reported by Neuberger & Edinger,¹² in which at the times of defoccation standstill of the ventricle occurred, resulting in repeated syncope. The case may probably be placed in the category of standstill of the whole heart, for at autopsy an aneurism was found upon the basilar artery, so situated that it was probable that with a sudden rise of blood-pressure, serious pressure would be exerted upon the medullary centres. Unfortunately the case was reported before the more modern graphic methods were available, and there is no certain evidence, but only strong presumption, of true standstill.

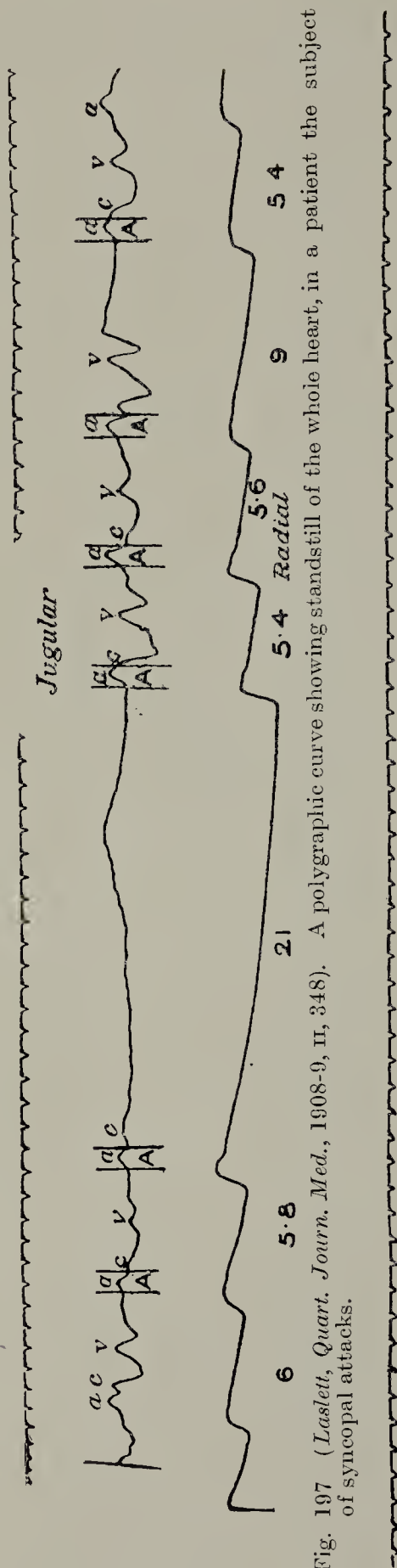


Fig. 197 (*Laslett, Quart. Journ. Med.*, 1908-9, II, 348). A polygraphic curve showing standstill of the whole heart, in a patient the subject of syncopal attacks.

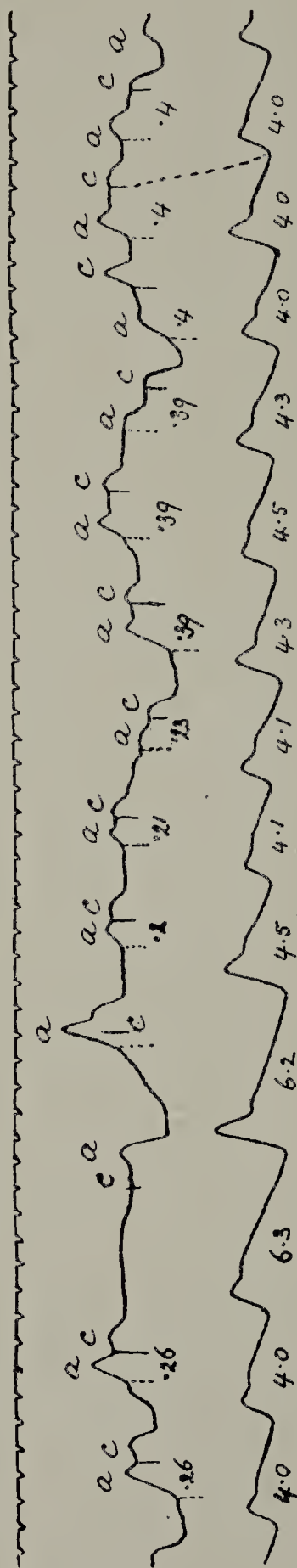


Fig. 198. (*Quart. Journ. Med.*, 1908-9, II, 358). A polygraphic curve from a case of mitral stenosis, showing the central portion of a phase of heart slowing of vagal origin. The ventricle escapes before the auricle after the first long pause, and shortly after the auricle at the end of the second long pause. Other and similar curves from this patient are shown in Fig. 87 and 89.

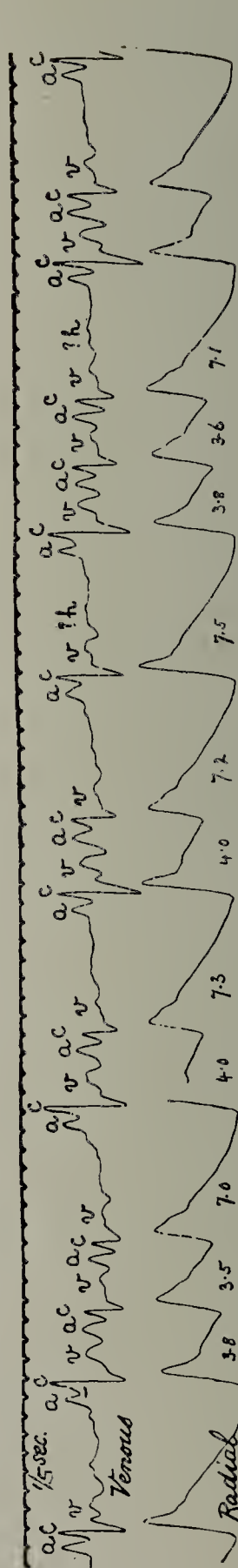


Fig. 199 ($\times \frac{1}{10}$) (*Heart*, 1909-10, I, 300). Polygraphic curve showing marked irregularity of the whole heart during a period of suspended respiration. Taken from the same case as Fig. 195, and therefore attributed to vagal influences.

In the recently recorded cases of Mackenzie¹¹ & Laslett,⁷ we have perfect examples of the phenomenon. In Mackenzie's case, mitral stenosis was present, and the patient was under digitalis; pauses as long as two and a half seconds occurred, during which no systole of auricle or ventricle took place. Laslett's case is an even more interesting instance. The patient, in whom no definite sign of cardiac disease could be found, manifested frequent standstill of the whole heart of four or five seconds duration, (Fig. 197), and these pauses in the action of the heart were often accompanied by syncopal seizures. Occasionally, longer pauses of six to eight seconds were also recorded. The pauses bore no relationship to respiration, but were abolished by the administration of a hypodermic dose of $\frac{1}{50}$ grain of atropine.

Periods of standstill of a very similar nature have been recorded by Wenckebach,²² who has spoken of them under the title of "Luciani's periods."

There are a number of cases in the less recent publications which cannot be passed over without reference. Thus Pope¹⁴ described a horse with a pulse of 10 per minute (the usual rate is 35-40), which suffered from syncopal attacks and which was found at the post-mortem to have "dropsy of the cervical region." Stackler¹⁷ described a patient whose pulse was commonly 40 to the minute and who was the subject of attacks in which still further retardation occurred. At the autopsy the right vagus was seen to be involved by an indurating tumour. These and many other examples, of which the more notable contain evidences of fracture of the upper cervical vertebrae, gross cerebral lesions, contractions of the *foramen magnum*,^{2 & 6} are often quoted but a detailed consideration of them would lead us far afield. The evidence in regard to many of them is so meagre and the issues so involved, that at the present time their further consideration would be unprofitable. Those who desire a closer familiarity with the subject may consult the collected cases of Edes.⁵ In discussing the vagus in relationship to derangement of the cardiac mechanism, it is sufficient to illustrate the separate varieties of disorder by means of a few of the more striking instances, and to confine the account especially to examples in which clear graphic records have been obtained. Sufficient parallels to experimental standstill are found in the cases of Laslett and Mackenzie.

Escape of the ventricle in man. As in the experimental so in the clinical condition the return of the rhythm of the ventricle from a condition of standstill may be heralded by one or more idio-ventricular contractions. Mackenzie's patient¹¹ may be cited as an example. Ventricular escape is also found in conjunction with the phasic variation of heart rate already described.⁸

Fig. 198 is from such a case, demonstrating ventricular escape at the end of long pauses (cp. Fig. 87 & 88).

Complex conditions. It is obvious that several of the characteristics shown by irregularities in the mechanism of the heart as a result of inhibitory

influences presented in the experimental animal and in man, may be combined in a single patient. Thus a marked respiratory arrhythmia may be associated with a periodic and phasic variation in a single case. An irregularity usually associated with breathing may be independent of it in the same case from time to time (Fig. 199). Further types of irregularity met with in the same patients, and probably of similar origin, are not infrequently of a high order of complexity. An example of such a disturbance is shown in the accompanying figure, (Fig. 200).

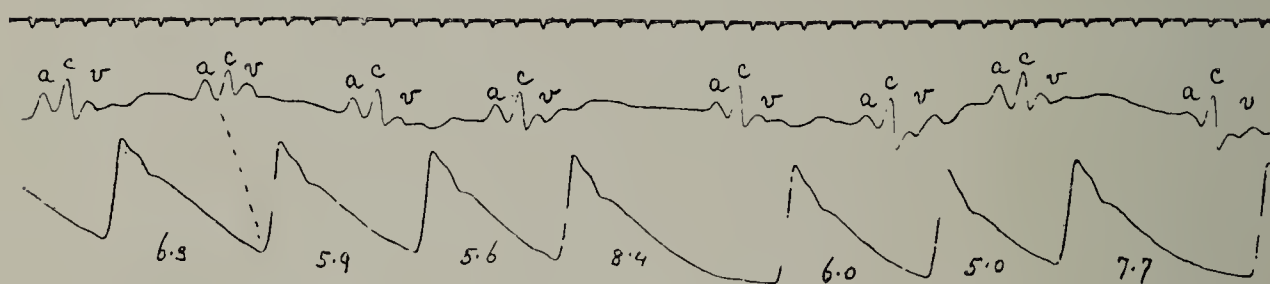


Fig. 200 ($\times \frac{5}{6.3}$) (Heart, 1909-10, I, 303). A polygraphic curve showing gross irregularity in the lengths of pulse intervals; the whole heart participated in the irregularity. From the same case as Fig. 191.

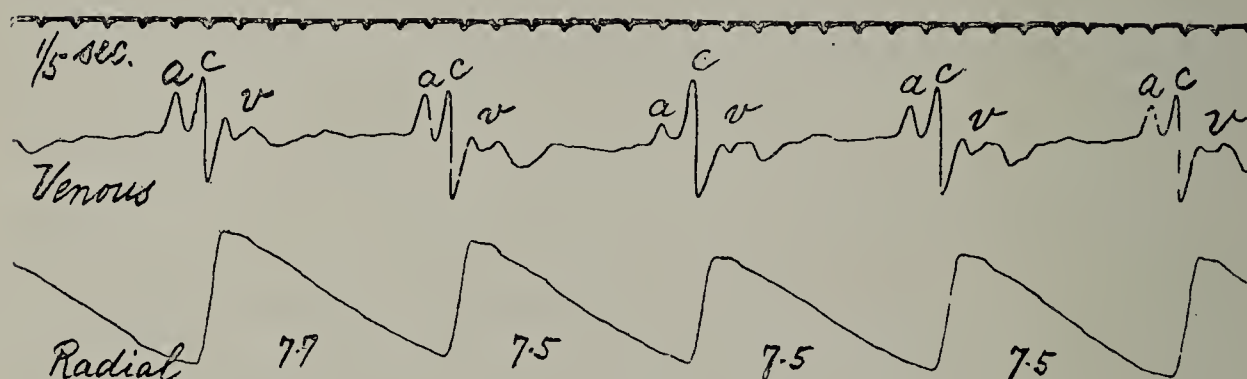


Fig. 201. (Heart 1909-10, I, 303). Polygraphic curve showing marked slowing of the whole heart; the rate is approximately 38 per minute. The slowing occurred in the case from which Fig. 200, etc., were taken and is therefore attributed to the vagus.

General remarks.

In the preceding pages, a number of examples of irregularity are given which may be definitely stated to be of vagal origin. And the phenomena cited are the only ones which we are completely justified in attributing to vagal influences at the present time. It has been customary in the past to assign many forms of irregularity of the heart to nervous influence, irregularities which are now known to originate independently of the central nervous system. It has been equally customary to attribute all forms of marked pulse or ventricular slowing to the vagus. The position has never been justified and it is now positively known that many such instances are due to local cause (for example, interference with the conducting tract). It is equally unsafe to attribute marked slowing

of the whole heart to this cause. Doubtless, many forms of slow pulse are manifestations of altered innervation, but their indiscriminate assignment to this category is not without serious risk of fallacy, and but very few examples of marked slowing have received detailed and complete study. It is not long since acceleration of the pulse was assigned to the withdrawal of vagal impulses, and this was named by Nothnagel¹³ as the general cause of paroxysmal tachycardia. We are now aware that paroxysms of tachycardia arise as a rule, not at the pace-maker but at some point removed from it. The tracings of the commencement and termination of such ectopic paroxysms are in themselves sufficient to exclude both vagal or sympathetic influence as their direct and provocative agents.

The chapter of our real knowledge of the vagus in its action upon the human heart closes at this point. It is suspected of influences in other disorders of the heart's mechanism. It has been alleged as a cause of the heart's failure in diphtheria,* it has been accused as a malefactor in angina pectoris and in many other conditions, but upon totally insufficient evidence to secure conviction.

It is still open to question how far it is through its influence that digitalis produces its characteristic slowing in cases of auricular fibrillation; and its connection with the fits of heart-block is still debatable. Some further remarks upon the last question are offered in the succeeding chapter.

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* In dying diphtheritic patients in which the heart is slow, examination by modern methods has discovered the presence of heart-block, (Fleming & Kennody, *Heart*, 1910-11, II, 77).

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CHAPTER XXII.

THE SO-CALLED "ADAMS-STOKES" SYNDROME."

The syndrome of Adams¹ & Stokes¹⁷ may be described as a clinical condition in which a persistently slow pulse is associated with syncopal or epileptic attacks.

In discussing certain pathological aspects of this syndrome it should be clearly understood at the outset that the definition includes several separate pathological entities. The majority of the typical cases are unquestionably the result of heart-block. But slow action of the ventricle is common apart from disturbed conduction, and attacks of faintness or momentary giddiness are readily included in the comprehensive term syncope. Marked slowing of the ventricle (to 30 or 40 per minute) in the absence of heart-block is rare, but it does occur and it may be accompanied by nervous manifestations. The writer has seen a patient in whom the sinus rate varied from 25-40 beats per minute, and in whom fits occurred. Cases of vagal slowing of the whole heart, such as are described in the previous chapter, have also to be borne in mind; cessation of the ventricular contractions at the time of the fits is in itself insufficient evidence of heart-block. There are also instances of marked slowing of the pulse as a result of the occurrence of early premature contractions, and the symptom may be combined with true epilepsy. A case of this nature has come to the writer's notice and similar cases have been reported by James^{10a} and Schmidt¹⁵. It should also be remembered that heart-block and fits are not necessarily accompanied by a persistently slow pulse.

Having emphasised the importance of distinguishing the several types of clinical cases, attention may be more especially devoted to the chief group of patients, those in whom the syndrome is the outcome of damage or destruction of the auriculo-ventricular bundle.

The cause of the syncopal and epileptic seizures.

In patients who suffer from heart-block, grave symptoms referable to the nervous system are not uncommon; they consist, according to the length of the attack, of transient attacks of giddiness or dimness of vision, momentary loss of consciousness, epileptic seizures and finally death. They are accompanied by slowing or standstill of the ventricle, and are directly attributable to these events (Fig. 202 and 203). It is the occurrence of the

preliminary lapse of the pulse beats first described by Webster,¹⁸ the definite relationship of the length of the pause to accompanying phenomena and finally the re-appearance of the pulse beats before the return to consciousness, which enable us to conclude that the pulse slowing is the actual cause of the fits, and that the loss of consciousness depends upon a derangement of nutrition consequent upon such slowing. We have in the numerous cases on record ample evidence of these time-relations; and the details reported allow of certain general statements. There is little or no disturbance provided that the ventricular stoppage lasts but two or two and a half seconds. In the majority of cases, cessation for three to five seconds results in momentary unconsciousness. An absence of ventricular contraction of fifteen to twenty seconds is accompanied by the commencement of epileptiform phenomena; an absence for ninety to one hundred and twenty seconds is rarely followed by recovery. These figures are given after an examination of a large number of published cases. They are not rigid, but are in general accordance with the mass of evidence.

Loss of consciousness and convulsive movements follow copious bleedings, whether experimental or therapeutic. The nervous symptoms are unquestionably due to anæmia of the brain; a position clearly substantiated by a number of experimental observations. Kussmaul and Tenner¹² compressed the carotid arteries in a number of male adults; dilatation of the pupils, slow deep and sighing respirations, dizziness and unconsciousness ensued. Schiff¹⁶ and Hill¹⁰ produced unilateral convulsions by compression of a single carotid.

We have in the retardation of the ventricular rate and the consequent cerebral anæmia an ample explanation of all the phenomena observed in the majority of the fits of heart-block.*

The cause of the ventricular slowing.

It will be perceived that in ascribing the nervous phenomena, purely and solely as we must, to interference with the vascular supply of the brain, an important step is made; for having ascribed them to this cause, we cannot look for further information from them to aid us in ascertaining the origin of the fit. The cause must be sought in the heart itself, and in its nervous connections.

In studying the fits, there is abundant evidence to show that in cases either of complete or incomplete block, the cause of the wider spacing of the ventricular contractions must be sought in a portion of the musculature distal to the auricle. It has been shown repeatedly^{2, 3, 4, 11 & 13} that the auricles continue to beat during the pauses.

* Occasionally the fits are not the result of ventricular stoppage, but are accompanied by an increase of ventricular rate; ^{9 & 18} under these circumstances the contractions are of abnormal type and are probably insufficient to maintain the cerebral circulation.

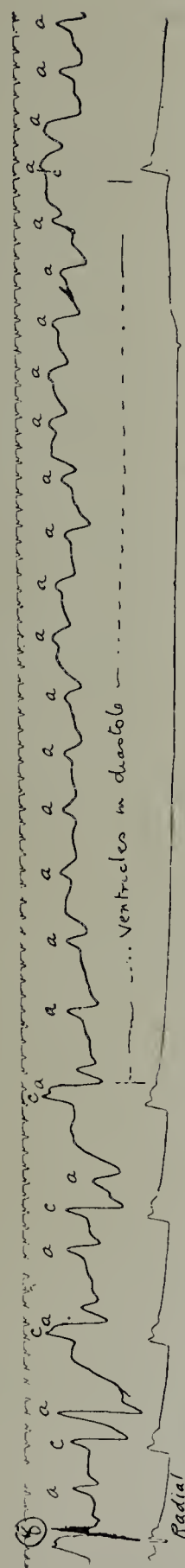


Fig. 202 ($\times \frac{1}{11}$). A polygraphic curve, kindly lent by Dr. John Hay. Taken from a case of Adams-Stokes' syndrome. The opening portions of the curve show complete dissociation. The ventricle suddenly ceased to beat for a period occupied by fifteen auricular cycles, a time interval of 11 seconds. The pause was accompanied by loss of consciousness, and was but one of a series of similar pauses, some shorter some much longer, observed in the same patient at the same time.

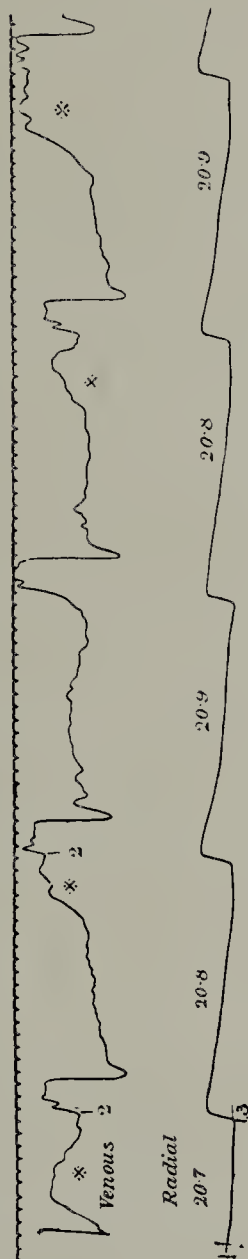


Fig. 203 ($\times \frac{1}{1}$) (Lewis and Mack, *Quart. Journ. Med.*, 1909-10, III, 273, Fig. 8). A polygraphic tracing taken from a case of auricular fibrillation and complete heart-block (Fig. 175-178 are from the same case). On this occasion the pulse slowed to about 14 beats per minute and the patient was on the brink of unconsciousness. The irregular waves marked with asterisks were due to the restlessness of the patient and are more marked towards the ends of the pauses. The venous pulse is of the ventricular form.

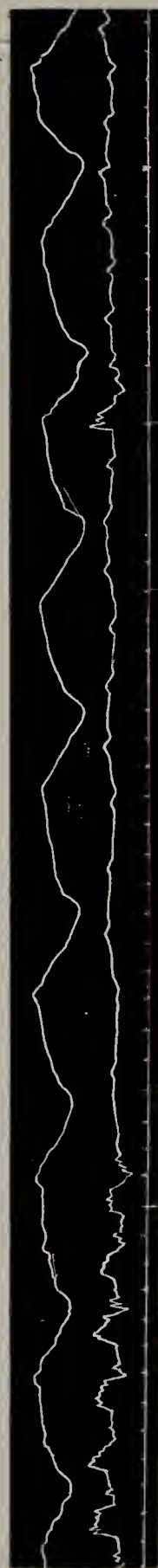


Fig. 204 (Erlanger and Blackman, *Heart*, 1909-10, I, 197). Respiratory and apical curves from a dog, five weeks after the auriculo-ventricular bundle had been crushed and its functional continuity destroyed. On the day upon which the curves were taken, the animal had syncopal attacks. The curve shows the termination of a series of rapid ventricular beats and "stoppage" of the ventricle. The auricles continue to beat.

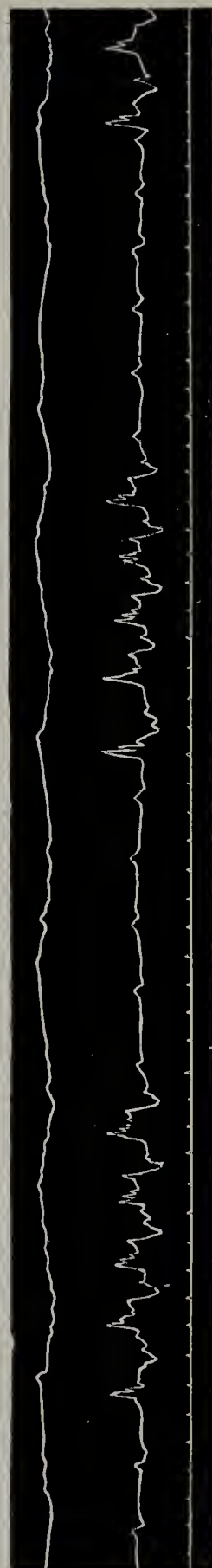


Fig. 205. (Erlanger and Blackman, *Heart*, 1909-10, I, 197). From the same animal, showing group beating of the ventricle and ventricular stoppage. The time marker in this and the preceding curve is in $\frac{1}{1}$ sec.

The fits of partial heart-block. It is now a generally recognised fact that fits are more frequent in partial than in complete heart-block. The observation of a slowing of the ventricle, in the absence of decrease in the rate of the auricle, indicates an increase in the grade of block. The fits of partial heart-block may consequently be attributed to this cause.¹³ An increase to a high grade of partial heart-block, 10 : 1, 20 : 1 ratios, etc., provides a clear solution of the ventricular slowing. It may be asked why under such circumstances the idio-ventricular rhythm fails to appear? The reply is that this rhythm is often dormant, though the exact conditions which govern its dormancy or awakening are not yet clear. It seems that the more rapidly the grade of heart-block is increased, the longer will be the delay in the appearance of spontaneous ventricular beats.⁵ But there are unquestionably other factors in operation, the exact nature of which is at present unknown to us. When we inquire as to the cause of the increase of block, it must be admitted that our knowledge is scanty. Two chief influences have been cited, and these have received brief discussion in other chapters, they are firstly, a sudden increase of auricular rate⁴ and secondly, vagal inhibition. Both have been suspected in individual cases, for either will produce stoppage of the ventricle experimentally; fits have been occasioned by deliberate compression of the vagus,¹³ but the usual provocative cause in patients is still unknown.

The fits of complete heart-block are less frequent, but a number of instances have been recorded, and many graphic records have been published.* The mechanism of production is obviously distinct from that of the attacks in partial dissociation. The vagus has been supposed to cause the ventricular slowing. It is known that the vagus exerts a direct inhibitory influence upon the ventricle in a large percentage of mammalian hearts, for its influence can be demonstrated upon a heart in which a heterogenetic rhythm is proceeding from the ventricle. In many instances, stimulation of the vagus inhibits the paroxysm (Fig. 206). But the experiments of Erlanger,⁶ Hering⁹ and Rihl,¹⁴ seem to show that in a heart in which the auriculo-ventricular bundle has been destroyed, vagal influences upon the ventricle are usually if not always removed. That is to say it appears that the majority, if not all, the inhibitory vagal fibres pass to the ventricle through the bundle.

The experiments of Erlanger & Blackman⁷ direct attention more especially to the ventricle itself. These workers have succeeded in reviving dogs after the bundle had been crushed and permanent dissociation had been

* Cases of complete heart-block seem more prone to enter a *status epilepticus*, although the fits as a whole are less frequent than in partial block, and it is in this state that most of the curves have been taken.

obtained. Several of the animals subsequently developed syncopal attacks similar to those of clinical heart-block, yet vagal stimulation in the same animals failed to effect the ventricular rate. The solution of the problem of the fits of complete heart-block rests with the investigation of the influences which increase or decrease the idio-ventricular rate, a rhythm which is known to originate in the junctional tissues (Chapter XI). The importance of the experimental work is twofold ; it has shown that a lesion of the bundle not only suffices to provoke dissociation, but that this simple lesion brings in its train, directly or indirectly, the nervous symptoms also ; secondly

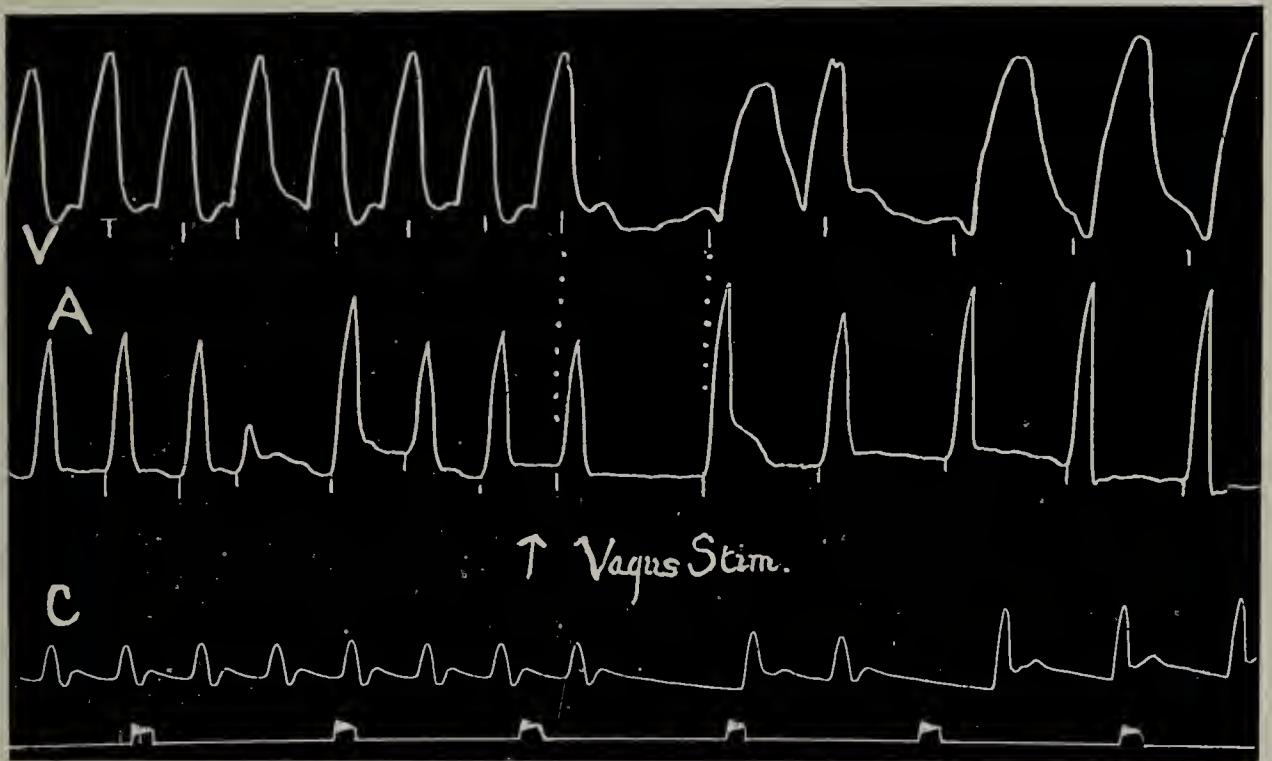


Fig. 206 (*Heart*, 1909-10, 1, 116). Myocardiographic curves (V—ventricle, A—auricle) and Hurthle carotid pressure curve (C) from a dog. Over the first half of the curve a ventricular tachycardia is present and the auricle is responding to it (shown by the single premature auricular contraction which disturbs the auricular rhythm only). Vagal stimulation inhibited the paroxysm. The vagus, therefore, has a direct action upon the ventricle. A single reversed beat, of a similar nature to the paroxysmal beats, interrupts the returning normal rhythm.

it has shown that the variations in the idio-ventricular rhythm, isolated from vagal influences, may be profound, not only in rate but in the sequence with which the beats follow each other (Fig. 204 and 205).

We may summarise the present position in regard to the fits of heart-block, by saying that in partial heart-block they depend upon alterations of conductivity ; that in complete dissociation, their appearance or non-appearance is consequent upon influences which affect the pace-maker of the ventricle. Finally, it should not be forgotten that the pace-maker of the ventricle lies in the immediate neighbourhood of the lesion which produces the dissociation,

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CHAPTER XXIII.

ALTERNATION.

Clinical Alternation.

IN 1972, Traube¹⁷ described under the term *pulsus alternans* a condition of the pulse in which large and small pulse beats follow each other in regular succession. He noticed in the example described that, while large and small beats were placed alternately throughout, the interval separating large and small beats exceeded that which separated small and large beats. The difference in intervals portrayed in Traube's figures was small, and in many examples which have been published subsequently it has been absent, that is to say, the incidence of the beats is perfectly regular. An illustration of the condition in which the intervals are uniform is given in Fig. 207a. The succession of pulse waves is perfectly rhythmic, and the irregularity consists in a variation in the height of the alternate primary waves.

In the earlier periods of its investigation this alternation in the arteriogram was confused with a regular bigeminy due to premature contractions,¹⁹ but the characters mentioned and the continuance of the condition for hours, weeks or months and under varying circumstances usually permit of its identification. When present in slight degree it is increased by exertion, by acceleration of the heart rate, and by the occurrence of premature contractions (Fig. 208). Its relationship to premature contractions is of very great interest and practical importance, for alternation may be confined to the few rhythmic pulse beats which follow a disturbance of an otherwise regular pulse, by a premature ventricular contraction. Under these circumstances the premature pulse beat and the pause which follows it are succeeded by a tall beat, and this in turn is followed by a short beat and then a taller beat.

The alternation in the height of pulse beats may terminate at this point or it may continue for a longer period. A premature contraction frequently serves, in this manner, to unmask a predisposition to *pulsus alternans* which would otherwise escape unobserved. Some curious and rare relationships of alternation to premature contractions have been reported by Windle, and are illustrated by Fig. 208 and 209. The alternation in the pulse generally

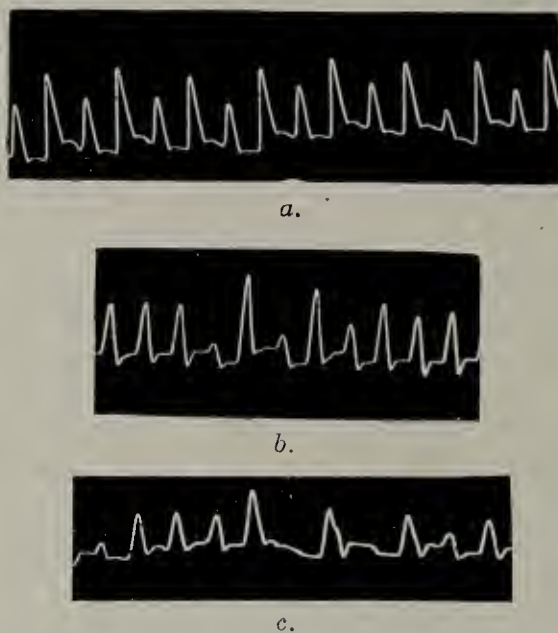


Fig. 207 (*Heart*, 1909-10, i, 49). Dudgeon curves taken from the radial artery of a patient during an attack of paroxysmal tachycardia; showing *pulsus alternans*.

- (a) Alternation of small and large rhythmic beats.
- (b) Alternation commencing with a small beat; note the corresponding exaggeration of the succeeding beat.
- (c) Alternation commencing with a large beat. The small beat which follows it scarcely affects the arterial curve.



Fig. 208. (*Windle, Heart*, 1910-11, ii, 96). Slight alternation is present in the first five beats of this radial curve. A premature contraction of the ventricle is followed by increased alternation. The second premature contraction is followed by a decrease of alternation.

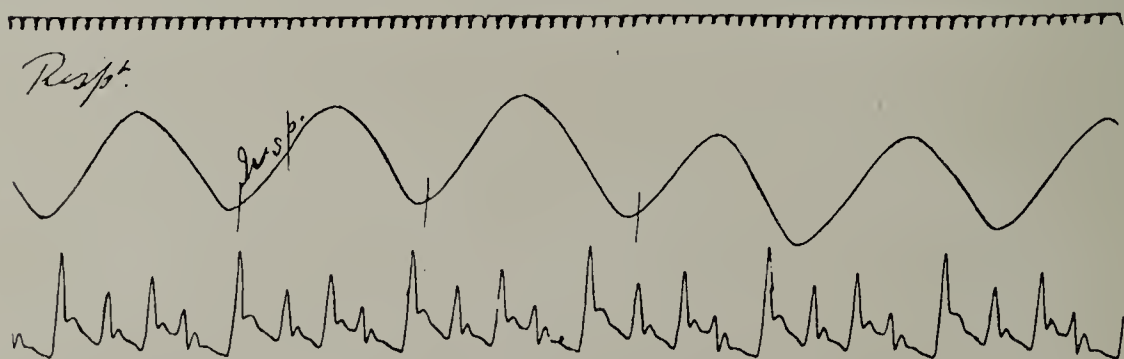


Fig. 209. (*Windle, Heart*, 1910-11, ii, 100). Respiratory and radial curves from a patient exhibiting a curious and regular mixture of *pulsus alternans* and premature contractions.

commences with the appearance of an unusually small beat (Fig. 207*b*), but occasionally it is seen to begin in a large beat (Fig. 207*c*). It varies greatly in its degree, alternate beats being at times almost imperceptible (Fig. 207*c*) and sometimes no trace of them is to be found in the arteriogram. Under the last named circumstance an apparent halving of the pulse rate occurs.

The mechanism of the heart while *pulsus alternans* is present has received considerable attention. It is known that the sequence of chamber contraction is normal, that is to say that each ventricular contraction is preceded by a regular auricular contraction. Cardiographic curves taken mechanically from the apex beat usually show a regular succession of alternating upstrokes. The alternation in cardiogram and arterial curve is generally *convergent*, that is to say, the large upstroke of the cardiogram corresponds to the large pulse beat, but as Hering¹ has shown, the two pictures may be *divergent*, the large cardiographic upstroke corresponding to the small arterial upstroke. But alternation may be present in one curve and absent in the other, while there may even be divergence of alternation between cardiograms obtained from different points on the same chest wall.⁴ The electrocardiographic curves, examples of which are shown in Fig. 210, 211 and 212, demonstrate many points of interest. The sequence of chamber contraction is shown to be normal; the ventricular complexes are of the physiological type; the ventricular contractions are of supraventricular origin, and the course of the contraction in the ventricular musculature is normal. The summits of the electrical variations frequently show alternation in height. But the large excursion of the pulse or of the ventricular mycardiograms may correspond to a tall or short *R* summit of the electric curve, and the tall *R* may be associated with a large, small or inverted *T*. Moreover, taking pulse beats on the one hand and the individual peaks of the electrocardiogram on the other, alternation may be present in one, while it is absent in the other; and the most curious variations from parallelism in the alternation to divergence in alternation are encountered in one and the same patient within the space of a few heart cycles, (Fig. 210-212).

Alternation in experiment.

A precisely similar phenomenon is encountered in experiment; and the clinical and experimental findings are so completely in accord in all their details that no doubt remains that the two conditions are identical. In experiments upon the intact heart, what appears to be an alternation in the degree of shortening of the strip which gives the mechanical record is not infrequently seen. Amongst the earlier records the beautiful example published by Gaskell³ in 1883 may be mentioned. The contractions of the ventricle are usually absolutely rhythmic; but there may be slight variations in the intervals, so that the smaller pulse beat is separated from the larger by a rather diminished interval; such examples are comparatively rare

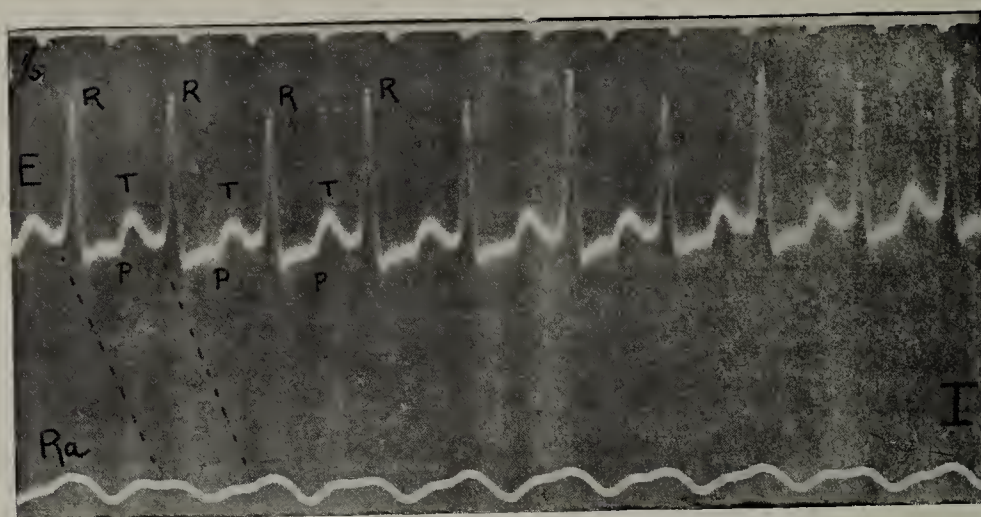


Fig. 210

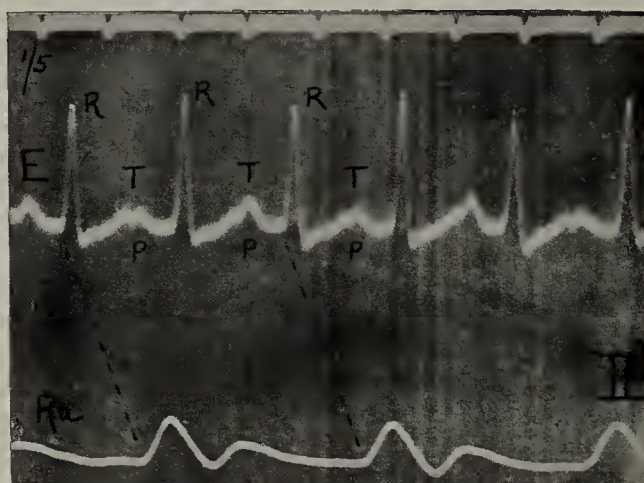


Fig. 211.

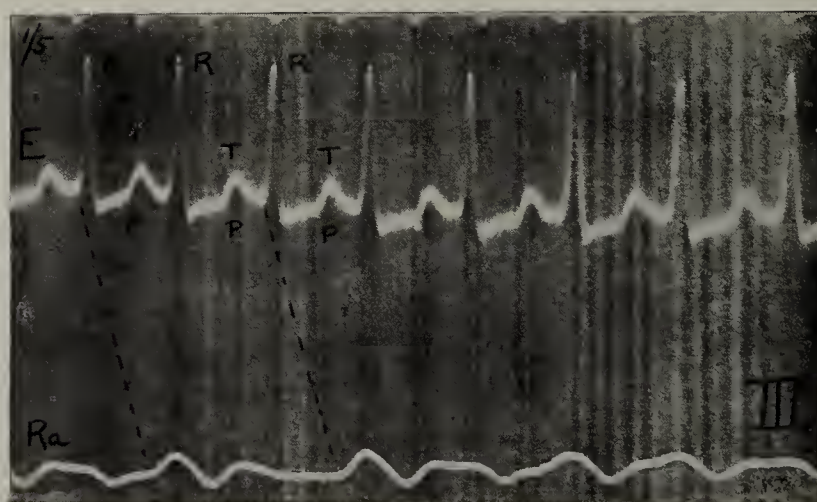


Fig. 212.

Fig. 210, 211 and 212 (*Quart. Journ. Med.*, 1910-11, iv, 141, *Fig. 3*). Simultaneous electrocardiograms and radial curves taken during paroxysms of tachycardia. From the same case as Fig. 215, etc.. Fig. 210 shows alternation in the electrocardiogram, but little or none in the radial curve. Fig. 211 shows alternation in both curves, in the radial curve it is extreme; the large *R* and *T* correspond to the absent radial pulsation. Fig. 212 shows alternation in the pulse and little or none in the electro-cardiogram.

(cp. Hoffmann,⁷ Fig. 10a). The variations in the degree of pulse change in experiment are similar to the clinical (Fig. 214); there may be but a slight difference in the height of alternate waves, or alternate ventricular contractions may completely fail to effect the arterial curves.¹⁶ Simultaneous curves from heart and artery may give divergent pictures, whether the former are taken from the intact chest wall⁴ or directly from the muscle (Fig. 213). In certain of the instances in which there is divergence between the alternation in ventricular and arterial curves, it can be shown that the divergence results from co-existing alternation in the force of auricular contractions¹⁰ (Fig. 213). The electrocardiographic curves are similar to the clinical and show the same curious relationships.^{5 & 9}

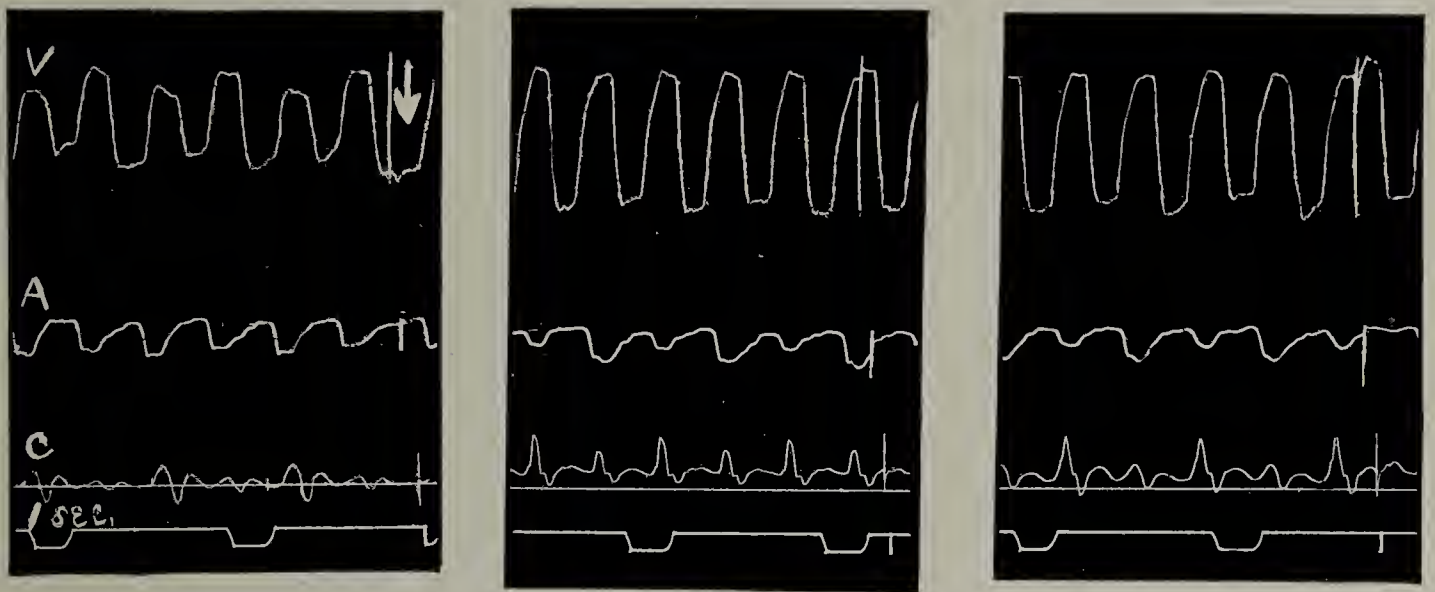


Fig. 213 (*Quart. Journ. Med.*, 1910-11, iv, 141, *Fig. 1*). Myocardiographic curves (*V*=ventricle, *A*=auricle) and carotid curve (*C*) from a dog's heart during an attack of tachycardia arising in the ventricle. The myocardiographic levers write downwards during systole. In the first curve the small ventricular beat corresponds to the small auricular beat and to the small carotid upstroke. In the second curve the small ventricular and auricular beats correspond to the large carotid upstroke. In the third curve the small ventricular and large auricular excursion correspond to the small carotid upstroke.

The length of alternate beats, estimated by myocardiograms, is often variable, the tall excursion accompanies the longer systole, and it is consequently followed by a shorter diastolic pause.² But it is questionable whether these tracings from the intact heart are entirely reliable in this respect. We know that premature ventricular contractions of the ventricle give rise to ventricular complexes which are of approximately the same length as the normal complexes; while in the myocardiogram, the premature contraction is usually conspicuously shortened. The electrocardiographic record usually shows them to be equal from one beat to the next, but in rarer instances the strong beat may be accompanied by a longer ventricular complex.⁷

The nature of alternation ; the conditions giving rise to it and its significance.

The similarity of the features displayed by clinical and experimental alternation is sufficient to indicate that we are dealing with one and the same heart mechanism in the two instances. Its nature has been widely discussed, but the facts in our possession are still too imperfect to permit the formation of final conclusions. The condition has been ascribed to heart-block in the ventricular walls,^{3, 4 & 14} The view has been entertained that it may be due to inaction of large areas of the ventricular musculature at alternate beats ; but this hypothesis appears to be precluded by an examination of the shape of the corresponding ventricular complexes in the electrocardiographic curves. It has also been attributed to alternation of contractility,^{6, 8 & 19} thus while it is supposed that the whole ventricle enters systole at each contraction of this chamber, the force of contraction is for some reason small or great at alternate beats. The chief difficulty, which hinders an acceptance of this theory, is the apparent inconsistency between the measured shortening of the ventricular muscle and the resultant wave in the arteriogram. The apparent inconsistencies between records taken from alternating hearts by one or another method form in fact some of the chief characteristics of the condition, and until more exhaustive study of the phenomena has been undertaken, no settled opinion of the nature of alternation itself can be entertained.

Alternation of the heart is at once one of the most mysterious and most important mechanisms of the heart with which clinicians have to deal ; and, treating it as a single phenomenon and in an empiric fashion, we may proceed to a brief description of the conditions in which it appears and to its significance. Studied in patients it is seen under two sets of circumstances. First, when an apparently healthy muscle is overtaxed, and notably when the heart is beating at an unusually high rate. It is a very frequent accompaniment of paroxysmal tachycardia. Secondly, it is found when the heart is beating within normal speed limits, but where there is reason to believe that the musculature is profoundly affected. It is particularly associated with senile changes of the heart muscle, and more especially with progressive fibrosis. It is frequently accompanied by grave symptoms, for example with pain of an anginal nature, and those who present it usually succumb, it is said,¹² within a few years. In experiment, alternation is also met with when the heart muscle is sound. It is often seen when the rate of contractions is increased, as the result of repeated stimulation, or where a paroxysm of tachycardia has been provoked by any means from a single point. It may be produced by the injection of digitalis¹ or antiarin¹⁶ (a closely allied body) into the circulation. Other poisons such as aconitine,² glyoxyl⁹ and hæmolytic serum bring about similar results.

Both in the patient and in experiment, when there is a predisposition to the condition, the tendency to its manifestation is increased by an additional call upon the heart, as for example, an increase of heart rate, or a

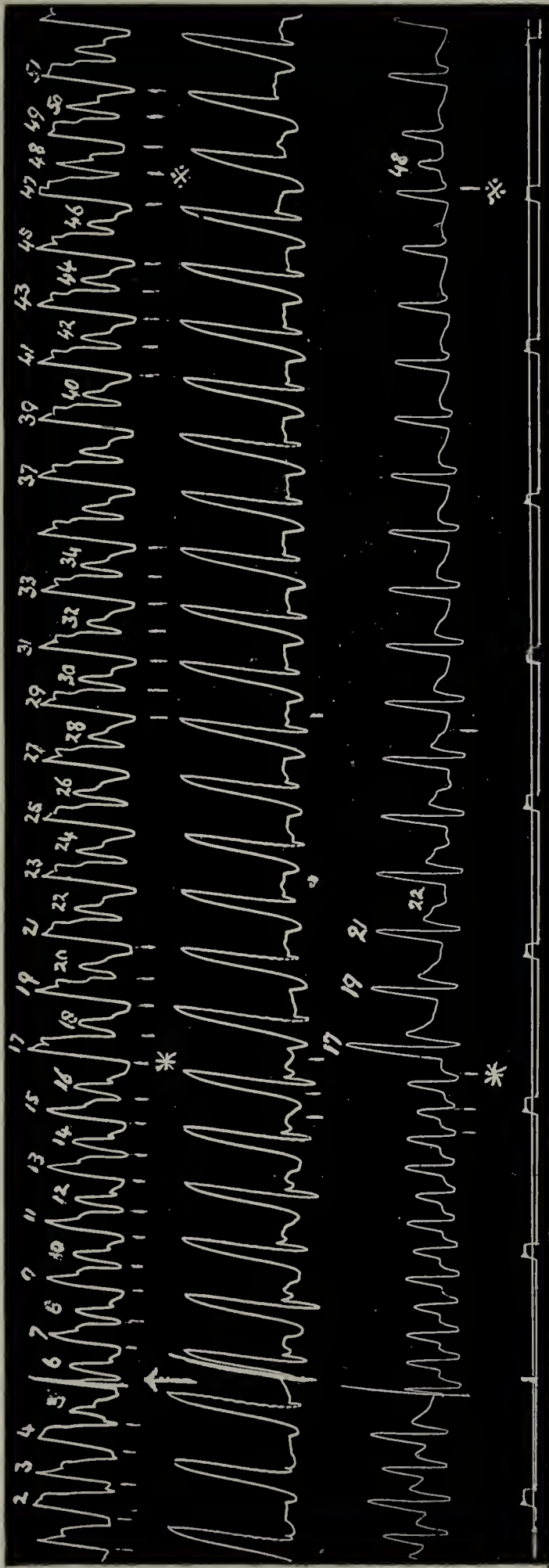


Fig. 214. Myocardiographic curves from a dog in which the right coronary artery had been tied and in which auricle (middle curve), is responding to each second beat of the ventricle (upper curve). The ventricle is alternating and interesting relationships are shown between the ventricular myocardiogram and the carotid curve (bottom line). Alternation to extinction is seen following beat 17. The change starts with a large beat (17) and an accompanying rise of mean blood pressure. Note the appearance of beat 48 in the carotid curve. The time is in seconds.

disturbance of rhythm by a premature contraction. Whenever it is seen in experiment or at the bedside, there is reason to believe, either that the heart muscle has been previously damaged, or that the heart is meeting an extraordinary demand for work. Alternation of the heart or pulse must be regarded therefore as of serious omen. It is only present when the muscle is in a precarious condition, be it structural or functional.

Yet alternation in itself is not of necessity an indication of incapacity; on the other hand, it might be regarded as the special response of muscle labouring to meet an exceptional task. That alternating action may be beneficial seems to be indicated from the fact that, when an abrupt change from a slight to an extreme grade of alternation takes place and the heart is beating rapidly, there may be an accompanying rise of blood pressure (Fig. 214).

In summing up this chapter it may be stated that at the present time we have knowledge of a definite, specific and readily recognisable mechanism, which is manifested both by experimental hearts and human hearts affected by disease, and that though we are as yet unaware of the exact means of its production, we are fully aware that it indicates an organ seriously embarrassed in its work, either on account of inherent defects in the organ itself, or on account of the stenuous effort which it is called upon to exert.

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INDEX AND DEFINITIONS.

INDEX AND DEFINITIONS.

<i>ABERRANT VENTRICULAR COMPLEX.</i> <i>A ventricular complex of unusual form associated with a contraction of the ventricle, which is due to a supra-ventricular impulse</i>		66, 67, 70, 140, 142, 180
A-C INTERVAL		13
Increase of		58, 92, 107
Measurement of		53
Shortening of		181, 235
ACONITINE		84, 276
ADAM-STOKES' SYNDROME		265
ALGEBRAIC SUMMATION OF ELECTRIC COMPLEXES		74, 76, 86
"ALL OR NOTHING LAW"		122
ALTERNATION		271
Aconitine and		276
Angina and		276
Clinical		271
Conditions giving rise to		276
Digitalis and		276
Electric curves in		273, 275
Experimental		273
Heart mechanism in		273
Myocardiograms in		275
Nature of		276
Pulse in ; (see <i>pulsus alternans</i>).		
ANATOMY		1
Morbid anatomy of bundle		99
Morbid anatomy in auricular fibrillation		232
Morbid anatomy in paroxysmal tachycardia		184
ANGINA AND ALTERNATION		276
<i>ANOMALOUS COMPLEXES.</i> <i>A complex (auricular or ventricular) which deviates markedly from the normal type for the same heart and same lead</i>		67, 139, 146
APEX CURVES IN HEART-BLOCK		90

ARTERIAL PULSE CURVES.

Alternation in	43, 271
Analysis of	43
Auricular fibrillation and	196, 223
Complete irregularity in	49, 196, 223
Heart-block and	90
Measurements from	51
Periods duplicated in	44, 45
Phasic variation in	46, 258
Premature contractions in	48, 49, 124, 128
Prominent beats in	44
Regular sequence of beats in	46
Respiratory variations in	43, 253
Spacing of	47

ASPHYXIA.

Heart-block in	84
<i>T</i> summit exaggerated in	74
" <i>As-Vs</i> " <i>INTERVAL</i> . <i>The time interval between the commencement of contraction of auricle and ventricle</i>	53
Increase of	58, 83, 92, 235
Reversal of; (see <i>Vs-As</i> interval).	
Shortening of	134, 182, 235
ATROPINE	110, 245, 261

AURICLE.

Direct leads from	36
Direction of contraction in	32, 36
Distension of	62, 199
Electric effects of contraction in (see auricular complex and <i>P</i> summit)	
Fibrillation of; (see auricular fibrillation)	
Hypertrophy of	72, 195
Pause following excitation of	41, 130
Point of primary negativity in	36
Premature contraction of	128
Tachycardia arising in	172

AURICULAR ELECTRIC COMPLEX. *The sum total of the electric variations or phases given by a single and complete contraction of the auricular tissues; (*P* summit)*

Absence of	207, 179
Anomalous	67, 139
Excited	32
Normal	23, 32, 66
Obscurity of	179
Variation of	32

AURICULAR FIBRILLATION. *A condition of the heart, in which the auricular musculature is in constant but inco-ordinate and ineffectual activity, and in which the normal impulses conveyed from the upper to the lower chamber are replaced by rapid and haphazard impulses.*

Arterial curves in	49, 196, 223
As a clinical entity.. .. .	228
Bigeminal pulse and	248
Clinical and experimental comparison of	226
Clinical	194
Complete irregularity and	207
Conduction of impulses and	247
Course of ventricular contraction wave in	212, 219
Diagnosis of	228
Digitalis and	242
Dissociation and	237
Duration of	229
Electric curves in	204, 212, 216, 218
Experimental	216
Heart-block and	235
Historical	194, 229
In the horse	230
Mechanism in	192
Mitral Stenosis and	107
Morbid anatomy in	232
Myocardiograms in	192
Partial heart-block and	239
Premature ventricular contractions and	247
Relation to tachycardia	190
Slow action of ventricle and	235-245
Vagus and	226, 239
Venous curves in	198, 201, 224

AURICULO-NODAL JUNCTION	6
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AURICULO-VENTRICULAR BUNDLE. *A specialised structure of the mammalian heart, by means of which the impulses promoting contraction are conveyed from auricle to ventricle, or from ventricle to auricle. (See bundle branches).*

Anatomy of	2, 7
Clinical lesions of	99-106
Discovery of	2
Experimental lesions of	80
Functions of	79, 113
In the rabbit	81
Partial section of	81, 83

DROPPED BEATS	83, 93
DYING HEART	40
<i>ECTOPIC AURICULAR CONTRACTION. A contraction which originates in some portion of the auricular tissue other than the pacemaker</i>	
	67, 139
<i>ECTOPIC BEATS. Beats which originate from impulses generated in areas of the heart musculature other than that of the pacemaker</i>	
	139
<i>ECTOPIC RHYTHM. A rhythm consisting of ectopic beats</i>	
	163, 176
ELECTROCARDIOGRAMS.	
Algebraic summation in	74, 76, 86
Alternation and	273, 275
Analysis of	65
Auricular fibrillation and	204, 212, 216, 218
Auricular and ventricular complexes in	23
Complete dissociation and	76, 86, 93
Constancy of	152
Dependence on direction of contraction.. .. .	26
Exophthalmic goitre and	74
Heart-block and	76, 86, 92
Heterogenetic rhythms and	155, 172
History of	18
Meaning of	25
Physiological forms of	21, 23
Physiological time-relationships	24
Premature auricular contractions and	139
Premature junctional contractions and	146, 154
Premature ventricular contractions and	146
Retrogression of contraction wave and	158
Value of	65
ELECTRIC VARIATIONS; (direction of)	25
EPILEPTIC SEIZURES	265
ESCAPE OF VENTRICLE	96, 97, 116, 118, 258, 261
EXOPHTHALMIC GOITRE; (electric curves in)	74
EXTRASYSTOLE; (see premature contraction).	
FIBRILLATION; (see auricular fibrillation and ventricular fibrillation).	
FITS IN HEART-BLOCK.	
Cause of syncope	265
Fits of complete heart-block	268
Fits of partial heart-block.. .. .	268
GALVANOMETERS	18
GLYOXYL	276

HEART-BLOCK. A condition in which there is partial or complete hindrance to the passage of impulses from auricle to ventricle.

Accelerated heart rate and	93, 234
Aconitine and	84
Adrenalin and	84
Alternating ratios in	93
Apex curves in	90
Arterial curves in	90
Asphyxial	84
Atropine and	108
Auricular fibrillation and	239
Chronic (experimental)	83
Clinical	89
Complete	83, 93
Degrees of	83, 92
Digitalis and	84, 108, 246
Dropped beats	83, 93
Electric curves in	76, 86, 92
Experimental	79
Factors influencing grade of	93, 235, 268
Fits of; (see fits in heart-block).							
Historical	79, 89
In the rabbit	81
Mitral stenosis and	107
Muscarine and	84
Oesophageal curves in	92
Partial	83, 93
Paroxysmal tachycardia and	234
Physostigmine and	84
Post-mortem findings in	99-106
Radiography in	89
Recovery from	84
Rheumatic infection and	107
Signs of	89
Swallowing and	111
Tachycardia and	93, 234
Unmasking of	235
Vagus and	108, 234, 268
Venous curves in	60, 90
Ventricular slowing in	266

HEMISYSTOLE. A term used to convey the idea of separate contraction of right and left ventricle

.. .. 148

HETEROGENETIC CONTRACTION.	A contraction arising in any portion of the heart, which has no essential place as an integer in a rhythmic series, and which is preceded by an interval of short duration.	(See premature contraction)	113, 122, 139
HETEROGENETIC RHYTHM.	A rhythmic series of heterogenetic contractions		120, 155, 172
Auricular			172
Experimental production of			156, 158
Nodal			181
Relation to nerves			158
Ventricular (clinical)			163
Ventricular (experimental)			158
HETEROGENETIC VENTRICULAR RHYTHM.			
Venous curves in			169
HOMOGENETIC CONTRACTION.	A contraction originating in a homogenous impulse		113, 117
HOMOGENETIC IMPULSE.	An impulse which belongs essentially to a rhythmic series, and which is comparatively slow in its process of formation		113, 117
HOMOGENETIC RHYTHMS			113, 120
H WAVE			204
IDIO-VENTRICULAR BEATS			96, 97, 116, 118
IDIO-VENTRICULAR RHYTHM.	The homogenetic rhythm of the ventricle; the intrinsic rhythm adopted by the ventricle when isolated from all extrinsic impulse formation		113
Origin of			113
Rate of			114
IMPULSE FORMATION; (rate of)			47, 114, 184
INTERFERENCES OF CONTRACTION WAVE			152
INTERPOLATED EXTRASYSTOLE			124
INTRA-AURICULAR PRESSURE			9
ISOELECTRIC AURICULAR COMPLEX			68
JUGULAR BULB			11
JUGULAR PULSE; :(see venous pulse).			
LEADS; (galvanometric)			21
MITRAL STENOSIS.			
Heart muscle in			107
P summit in			72
NEGATIVITY OF MUSCLE			26

PREMATURE CONTRACTION. *A contraction of any portion of the musculature of the heart before the anticipated instant; that is to say, before the instant at which such contraction would fall in continuing a regular succession of beats.*

Cause of	126
Origin in musculature	126
Paroxysmal tachycardia and	167, 176, 190
<i>Pulsus alternans</i> and	44, 271
Relation to nerves	126

PREMATURE AURICULAR CONTRACTIONS. *A premature contraction arising in the auricle*

Blocked	69
Electric curves and	139
Mechanism of	129
Myocardiograms and	129
Recognition of in man	132
Venous curves and	60, 132

PREMATURE JUNCTIONAL CONTRACTIONS. *A premature contraction arising in the junctional tissues*

Electric curves and	146, 154
Mechanism of	132
Myocardiograms and	135
Recognition of in man	136
Venous curves and	59, 132

PREMATURE NODAL BEATS; (see premature junctional beats).

PREMATURE SINUS CONTRACTIONS 130

PREMATURE VENTRICULAR CONTRACTION. *A premature contraction arising in the ventricle.*

Arterial curves and	49, 124, 128
Auricular fibrillation and	247
Heart-block and	114
Electric curves and	146
Interpolated	124
Location of	148, 152
Mechanism of	122
Myocardiograms and	122
Recognition of in man	126
Venous curves and	128

PRIMUM MOVENS; (see pacemaker).

P-R INTERVAL	24, 53
Prolongation of	86, 92
Shortening of	182
Variations in	86

PROMINENT VENOUS PEAKS	56
P SUMMIT; (see auricular electric complex)	23
PULSE; (see arterial pulse curves).	
PULSUS ALTERNANS	43, 271
Premature contractions and	271
PULSUS IRREGULARIS PERPETUUS	195
PURKINJE FIBRES	3, 7
Q SUMMIT	24, 82
REFRACTORY PERIOD. <i>The period of a heart-chamber-cycle during which its muscle refuses to respond to artificial excitations</i>	122
RELATIVE COMPLETE HEART-BLOCK. <i>A condition in which, though the junctional tissues are capable of transmitting impulses, few or none are actually transmitted on account of the presence of an idio-ventricular rhythm</i>	116
RESPIRATORY ARRHYTHMIAS	253, 254
RETROGRADE CONTRACTIONS	134
RETROGRESSION OF CONTRACTION WAVE	134, 166
Electric curves in	158
REVERSED HEART BLOCK. <i>A condition in which there is an increased difficulty to the passage of impulses from ventricle to auricle</i> 158, 160 (footnote)	
R SUMMIT	23, 27
In fibrillation	205, 219
Variations in	142
SINO-AURICULAR NODE. <i>A specialised interlacement of fine muscle fibres and nerve elements, buried beneath the superior end of the sulcus terminalis of the mammalian heart. The heart beat probably originates in it</i>	1
Anatomy of	2
Excision or section of	40
Freezing and clamping of	41
Function of	37
Measurements of	39
SINUS BEATS; ("sinus extrasystoles")	130
SINUS; (warming and cooling of)	40
"SPACING" (of arterial curves)	47
SQUILLS	108, 245
S SUMMIT	23, 28
Variations in	28, 142
"STANDSTILL." <i>The abrupt occurrence of an unusually long pause affecting the whole heart.</i>	
Clinical	259
Experimental	258
STANNIUS LIGATURE	31
STIMULUS PRODUCTION; (rate of)	47, 114, 184

VENOUS CURVES	51
Analysis of	55
Auricular fibrillation and	198, 201, 224
Fibrillation waves in	204, 226
Heart-block and	60, 90
Paroxysmal tachycardia and	169, 172
Premature contractions and	60, 128, 132
Prominent peaks in.. .. .	56
Synchrononous contraction of auricle and ventricle and	56, 62, 136

VENOUS PULSE.

Analysis of	51
Historical	9
In animals	10
In man	10
Mechanism of	13
Method of recording	11
Stasis wave	204
Time-relations	13
Variation in outline	199
Ventricular engorgement and	62, 199
Ventricular form of	62, 186, 198, 224
Waves of	13, 201, 204

VENTRICLE.

Premature contraction of	122
Tachycardia arising in	155

VENTRICULAR ELECTRIC COMPLEX. The sum total of the electric variations or phases given by a single and complete contraction of the ventricular tissues.

Aberrant	66, 67, 70, 140 142, 180
Anomalous	67, 139, 146
In auricular fibrillation	212, 219
In dissociation	76, 113
In paroxysmal tachycardia	163, 180
Of supraventricular origin	65

VENTRICULAR ESCAPE	96, 97, 116, 118, 258, 261
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VENTRICULAR FIBRILLATION	160, 188
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VENTRICULAR FORM OF VENOUS PULSE. A form of venous pulsation in which all the constant and prominent waves fall within the confines of ventricular systole

62, 186, 198, 224

" *V_s-A_s* " *INTERVAL.* *The time interval between the commencement of a ventricular and the succeeding auricular systole. It is of significance where the sequence of chamber contraction is reversed* 134

V WAVE.

Cause of	15
Fixation of	53
Time-relations of	13
Value of	53
<i>X DEPRESSION</i>	16
<i>X₁ DEPRESSION</i>	16
<i>Y DEPRESSION</i>	16
<i>YOUTHFUL IRREGULARITY</i>	254



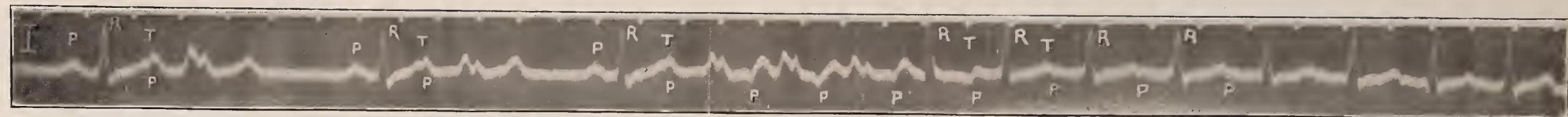


Plate I. Fig. 215 ($\times \frac{2}{3}$). Three electrocardiograms from a case of paroxysmal tachycardia arising ectopically in the auricle. Fig. 113 and 175, taken from the same case, should be compared with these strips.

I. The onset of a paroxysm. At first there is a bigeminy of premature auricular contractions (the ventricular complexes accompanying them are of an aberrant form, cp. Fig. 113). The paroxysm opens with three beats of the same type and then proceeds with ventricular complexes of a form more readily recognised as supraventricular. The anomalous *P* variations (ectopic contractions) are recognised by the deformation which they produce in preceding *T* summits; sometimes they notch the centre, sometimes the commencement of *T*. The transition from one form of notching to the other is well seen in strips *II* and *III*, which are continuous curves from the centre of a paroxysm. In *II* the notch falls in the middle of *T*, it falls a little further back towards the end of the figure, and at the end of strip *III* is at the beginning of *T*, (cp. Fig. 49).

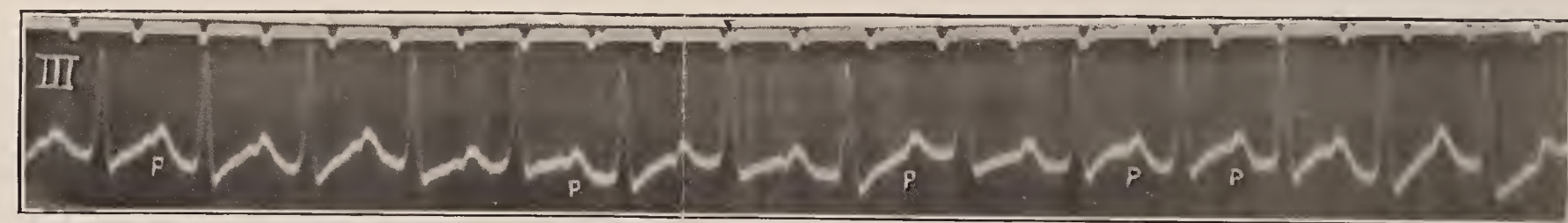
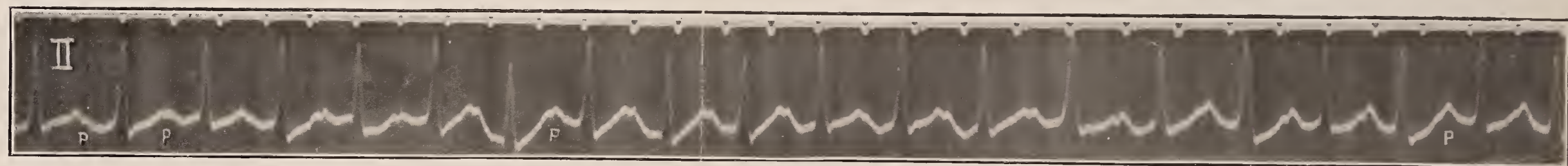


Fig. 215.

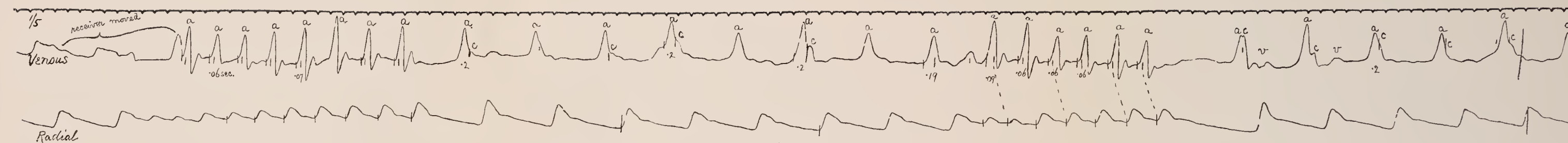


Fig. 216.

Plato II. Fig. 216. (*Heart*, 1909-10, 1, 306, *Fig.* 27). Venous and radial curves from a case of paroxysmal tachycardia. Two complete paroxysms are shown. The *a-c* interval during the slow periods is .2 sec. During the paroxysms the *a* wave is conspicuous and the calculated interval between it and the *c* wave is about .06 sec.

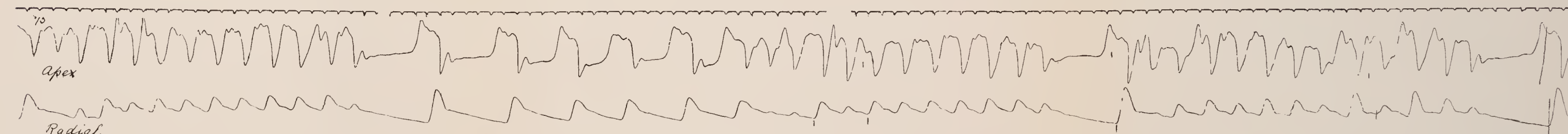


Fig. 217.

Fig. 217. (*ibid*, Fig. 28). Apex and radial curves from the same case, showing three paroxysms. In this and the preceding figure the paroxysms end in long pauses and there is some subsequent pulse quickening. The third paroxysm of the second figure is interrupted by two premature ventricular beats (as shown electrocardiographically).

Fig. 218. (*Hurt*, 1909-10, 1, 306, *Fig.* 26). Electrocardiogram and radial curve from a case of paroxysmal tachycardia (cp. Fig. 209 and 210). The curve shows a single complete paroxysm. In the electrocardiogram three normal beats, each accompanied by *P*, *R* and *T* waves are shown. They are succeeded by a paroxysm in which *R* & *T* waves of the same form appear. The ventricular complexes of the paroxysm are precoded by *P* summits, which are inverted, and the *P-R* intervals are reduced from .14 sec. to .08 sec. The paroxysm ends in a long pause, which is succeeded by three ectopic beats, the last of which is of the paroxysmal type, and finally by two normal beats.

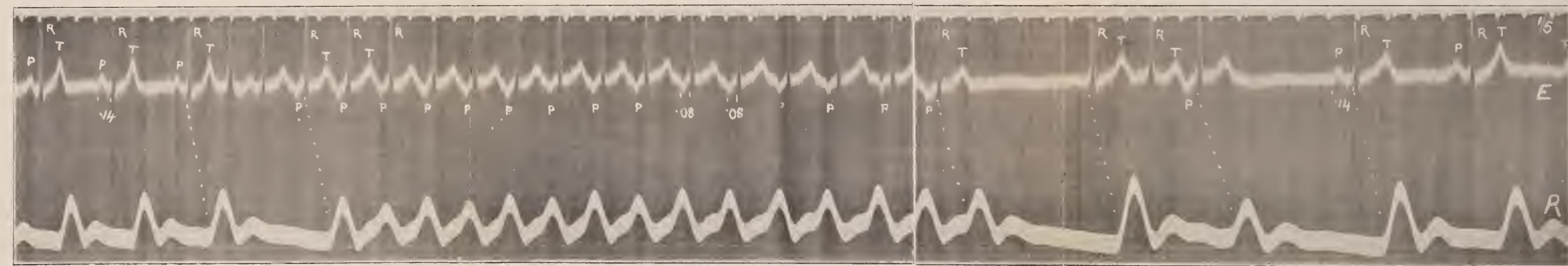


Fig. 218.

Plate III. Fig. 219 (*Quart. Journ. Med.*, 1910-11, iv, 145, *Fig. 4*). Cat. Chloroformed, and curarized. Vagi cut. An electrocardiographic curve, taken 3 min. 20 sec. after the onset of asphyxia and establishment of heart-block. The stimulation of the auricle and production of tachycardia, during a period of 2:1 heart-block, preceded the record and is not shown; the effects are manifested up to the point where the second ventricular beat of the figure occurs. The first and probably the second ventricular beats are idio-ventricular. The last three cycles belong to a 2:1 period; they belong to the period of recovery. The first two ventricular beats are considered to be idio-ventricular on account of the slight variation of the ventricular complexes from the normal type, and the relationship of the first *R* to its preceding *P*. Thus an auricular tachycardia converts a 2:1 heart-block into complete dissociation.

Fig. 220 ($\times \frac{9}{13.5}$). (*Quart. Journ. Med.*, 1910-11, iv, 145, *Fig. 2*). Decapitated Cat. 1 min. 45 sec. after the onset of asphyxia. A curve taken during the phase of prolongation of the *P-R* interval and showing the effect of interrupted induction shocks thrown into the auricular tissue. The prolonged *P-R* interval shows a further increase in the early phases of stimulation, and at the point marked in the curve passes into a 2:1 rhythm; at a further point marked in the curve dissociation appears. Complete recovery to a slightly prolonged *P-R* follows the cessation of stimulation.

Fig. 221 ($\times \frac{1}{2}$) (*Brit. med. Journ.*, 1910, ii, 1671). A continuous electro-cardiographic curve, in two sections, taken from a cat during a period of asphyxia. The curve starts when 2:1 heart-block was established; the auricle is represented by the variations *P*: the ventricle is represented by the variations *R* and *T*. At the point marked by the arrow in the upper curve the auricle was faradized, and fibrillation ensued. The type of curve immediately changes. The normal auricular waves are replaced by the fibrillation waves (fff). At the same time, though the ventricle shows no marked change of rate, its rhythm becomes irregular. The curve is continued, and shows the breakback from fibrillation + heart-block to a rhythm in which 2:1 heart-block is present. At the end of the lower curve the auricle was once more faradized and fibrillation ensued. The curves show the effect of heart-block in hindering the transmission of impulses from a fibrillating auricle.

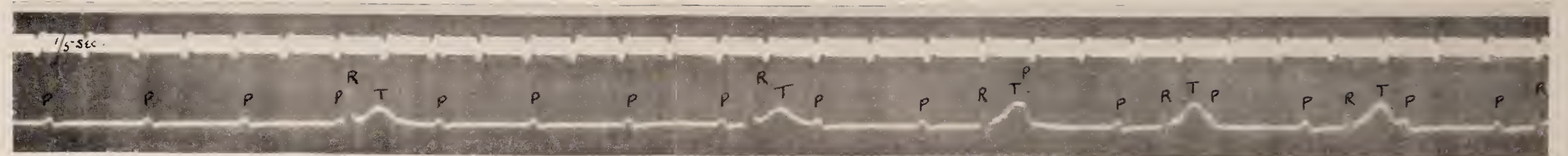


Fig. 219.

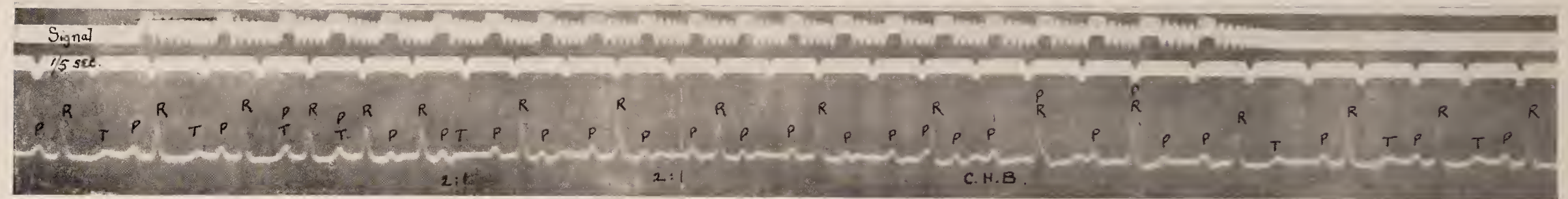


Fig. 220.

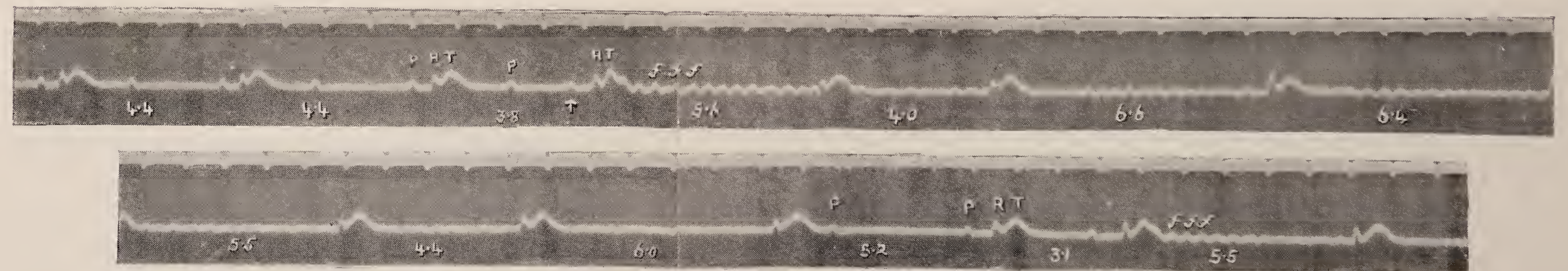


Fig. 221.

Plate IV. Fig. 222 ($\times \frac{3}{2}$). (*Heart*, 1909-10, I, 306, *Fig. 24*). Electrocardiographic and radial curves from a case of complete irregularity which exhibited the ventricular form of venous pulse. The electric curve consists of beats the result of supraventricular impulse formation (*R*, *T*), of anomalous complexes (the result of heterogenetic impulse formation in the ventricle (*En*)) and fibrillatory oscillations (*f*, *f*).

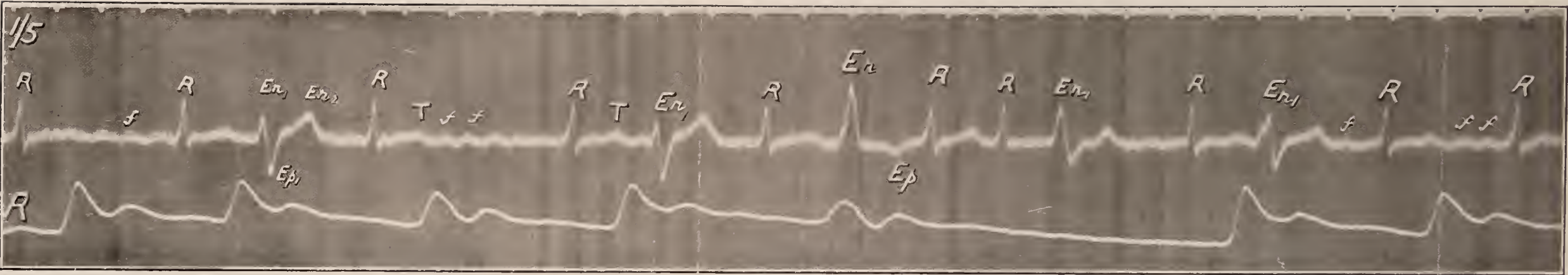


Fig. 222.

Fig. 223 ($\times \frac{1}{2}$). (*Quart. Journ. Med.*, 1909-10, III, 269, *Fig. 1*). Electrocardiogram from a case of complete heart-block and ventricular bigeminy. Showing that the second beat of each cycle is a premature contraction of ventricular origin. The curve consists of small ventricular complexes (*R*), which belong to the rhythmic ventricular beats, variations (*P*) scattered uniformly in the curve, and anomalous complexes, of the apical and left ventricular type, each coupled to a preceding rhythmic beat.

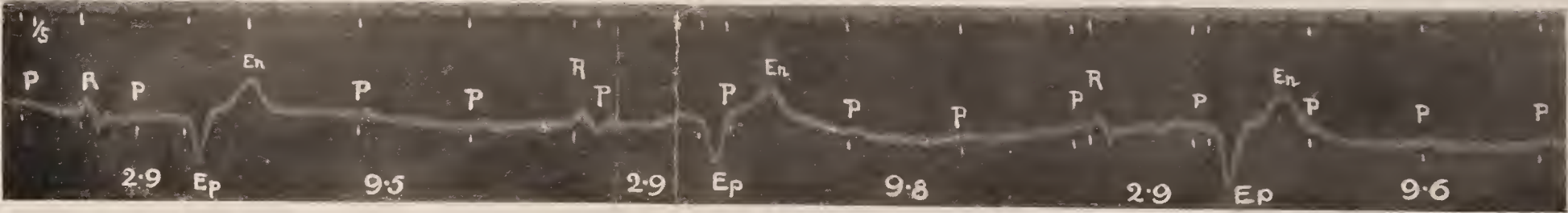


Fig. 223.

Fig. 224 ($\times \frac{3}{2}$). (*Quart. Journ. Med.*, 1909-10, III, *Fig. 1*). Polygraphic curve from a case of mitral stenosis in which auricular fibrillation was present. The venous pulse was of the ventricular form. The first six beats belong to the uncomplicated irregularity. From this point ventricular bigeminy is present until near the end of the curve. The bigeminy arose as a result of digitalis administration.

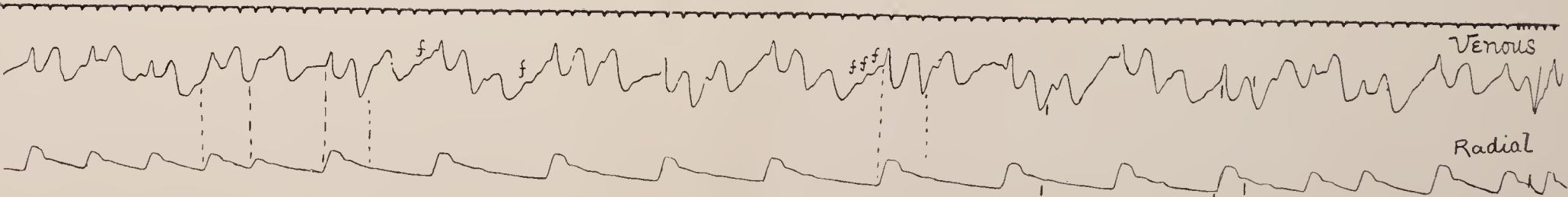


Fig. 224.

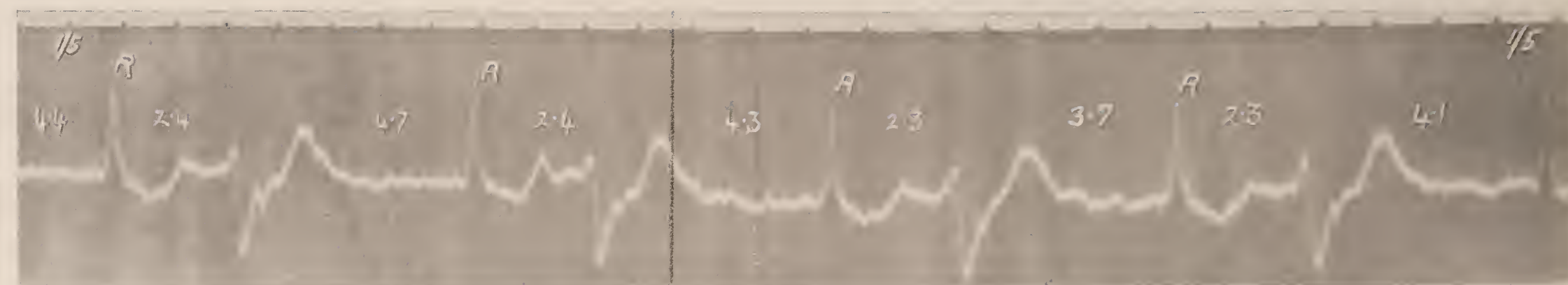


Fig. 225.

Plate V. Fig. 225, 226 and 227 ($\times \frac{2}{3}$). (*Quart. Journ. Med.*, 1909-10, III, 337, Fig. 2, 3 and 4). Three electrocardiograms showing the mechanism of coupled beats associated with complete irregularity of the heart (auricular fibrillation). From the same case as Fig. 224.

Fig. 225. The curve consists of the usual supraventricular responses *R*, *R*, and of anomalous complexes coupled to the former at a constant time distance $\frac{2.3}{5}$ sec., and of irregular oscillations, the result of the fibrillation in the auricle. The anomalous ventricular complexes are recognised as due to heterogenetic beats arising in the ventricle; they are followed by pauses of varying lengths.



Fig. 226.

Fig. 226 and 227. From the same case as Fig. 158. Two electrocardiograms with radial tracings. Mitral stenosis, auricular fibrillation and bigeminy on digitalis. They show similar events to Fig. 225. They were from an almost continuous curve. Note the maintenance of the accurate coupling despite the alteration in the type of the anomalous complexes.



Fig. 227.

